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OBSERVATIONS OF SERUM POTASSIUM IN PATIENTS UNDERGOING CARDIOPULMONARY BYPASS

by

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A THESIS
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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "Observations of Serum Potassium in Patients Undergoing Cardiopulmonary Bypass", submitted by John Robert Lampard in partial fulfilment of the requirements for the degree of Master of Science (Surgery).

ABSTRACT

Since Nernst (89) theorized that the electrochemical potential of the cell membrane resulted from the difference in the potassium concentration across it, considerable interest has been shown in the determination of the intracellular and extracellular potassium concentrations. The recent introduction of cardiac surgery (76) and the extensive use of drugs which affect the potassium balance in the body have re-emphasized the importance of differentiating the arrhythmias which arise from abnormal potassium metabolism and those which arise from other causes.

A study of twenty-six cardiopulmonary operations was made to identify the factors which influence the serum potassium concentration during surgery. Measurements were made of the acid-base balance, the oxygen consumption, the fluid balance and the urine formation. Results were compared with the serum potassium concentration during surgery. The effects of the preoperative medication, the duration of perfusion and the bypass body temperature on the serum potassium pattern during surgery were also noted.

The serum potassium pattern during surgery was found to increase during the prebypass period, rise slightly during bypass and drop to preoperative levels

during the postbypass and postoperative periods. Factors which influenced this pattern were: (1) the systemic blood pressure during prebypass, (2) the duration of perfusion, and (3) the amount of urine formed during surgery. Factors which did not influence the serum potassium were: (1) the preoperative medication, (2) the pH, (3) the oxygen consumption,and (4) the body temperature during bypass.

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patients in order to outline the causes of the serum potassium fluctuations during cardiopulmonary surgery.

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CHAPTER I

INTRODUCTION

Fifteen years ago Gibbon (76) performed the first surgical repair of the myocardium under direct vision. This advancement, made possible by the development of the artificial heart-lung machines, initiated surgical treatment for patients with congenital and acquired heart disease. It also introduced physical trauma to the heart along with artificial control of the respiratory and cardiovascular systems. Measurement of the effect of this insult have indicated that the interval required for the body to reverse the negative nitrogen balance may last as long as ten days (55, 80).

At the same time there has been a marked increase in the incidence of operative and postoperative arrhythmias (95). The use of digitalis to treat these arrhythmias has been complicated by the unpredictable washout of the drug that occurs during surgery (28). When coupled with the likelihood of a low operative or postoperative serum potassium concentration, the possibility of initiating an arrhythmia instead of abolishing it becomes extremely great.

Recent studies which measure the total body potassium deficit in the congestive heart failure patient (70, 85) have implicated the transmembrane potassium gradient

as one of the important causes of the increased incidence of arrhythmias. This has re-emphasized the need to identify those arrhythmias which arise from abnormal potassium metabolism from those which arise from digitalis or other causes.

In order to isolate the non-traumatic factors which influence serum potassium in patients undergoing cardiopulmonary bypass, the role of potassium in the metabolic response to the trauma will be outlined.

The Metabolic Response to Trauma

The central event in the injury response is the activation of the ANS integrating center, the hypothalamus (37). Activation of the hypothalamus occurs with emotional disturbances, operative premedication, general anesthetics, as well as tissue trauma (110).

Emotional reactions, particularly fear and apprehension, are potent stimulators of the hypothalamus and result in the elevation of plasma 17-hydroxycorticoid levels (110). The degree of preoperative anxiety can be measured in this manner.

Of the commonly used operative premedications (110), Morphine has a minor inhibitory effect on the ANS outflow while Demerol requires further study. Thiopental does not stimulate adrenocorticotrophic hormone (ACTH) or block the metabolic response to trauma. Ether anesthesia, through

ACTH release, has long been known as a potent stimulator of the adrenal cortex and has been used as an experimental stress model. However, part of its effect may be the result of hepatic and renal depression.

Halothane on the other hand, blocks sympathetic outflow and minimizes the action of norepinephrine on the heart and blood vessels, although this effect varies markedly with the duration and depth of anesthesia(94). As a general anesthesic it depresses the excitatory activity of the RAS and dampens the afferent impulses as they pass from the site of injury to the hypothalamus(94).

Blunt trauma and surgical trauma during general anesthesia, activate somatic receptor cells in a similar fashion. However, spinal anesthesia or paraplegia above the level of trauma blocks impulse transmission to the hypothalamus and hence, any neuroendocrine mediated metabolic changes (110).

Centers for three different efferent pathways have been found within the hypothalamus. These pathways stimulate the sympathetic outflow tract, the pituitary-adrenocortical system and the hypothalamoneurohypophyseal system to release catecholamines, ACTH and antidiuretic hormone (ADH). Efforts to study the metabolic response to trauma have been complicated by the intimate anatomical proximity and the functional interrelationships of these three systems (37). High plasma levels of catecholamines

release ADH(110). High concentrations of ADH can release ACTH (73). The catecholamine concentration required to release ACTH is probably never reached under physiological conditions (98).

Stimulation of the sympathetic outflow tract releases epinephrine from the adrenal medulla and norepinephrine from the post-ganglionic adrenergic nerve fibers. Epinephrine in turn alters body metabolism by elevating the plasma levels of glucose, lactic acid and free fatty acid (38). By activating the intracellular enzyme phosphorylase from the 'b' to the 'a' form (107), epinephrine depolymerizes glycogen into glucose-6-phosphate. Glucose-6-phosphatase, which is present only in liver cells, dephosphorylates the glucose-6-phosphate into glucose and phosphate which are then released into the hepatic vein(24). As the glucose leaves the cell in a manner not yet understood, it carries potassium with it (17). The potassium released by the liver is absorbed by muscle tissue by activation of the adenosine triphosphate (ATP) system in the cell membrane (31). Because muscle tissue lacks glucose-6-phosphatase, glucose is not formed, and the rising glucose-6-phosphate concentration stimulates the glucose anaerobic pathway inside the cell to form pyruvate and lactate which then pass extracellularly (23). Following the three to five minute catecholamine action, there is a prolonged hypokalemic phase during which time potassium

leaves the muscle cells and is transferred back to the liver (44).

Stimulation of the pituitary-adrenocortical system by hypothalmic corticotrophin releasing factor, (CRF) releases anterior pituitary ACTH (99). Upon reaching the adrenal cortex, the ACTH stimulates biosynthesis and release of glucorticoids and aldosterone(67). Although increased glucorticoid levels can be measured within two minutes of an ACTH injection, none of their metabolic effects can be demonstrated for several hours after injury (96). Hydrocortisone affects all stages of protein and amino acid mobilization and liver gluconeogenesis (71). Glucose production is markedly increased although glucose utilization is unaffected (71). The catabolic action of hydrocortisone mobilizes protein and potassium from tissue stores (39). Any diuretic effect which excretes the mobilized potassium is minor when compared with the effects of aldosterone (88). Aldosterone, the primary mineralocorticoid reduces sodium excretion and markedly increases potassium secretion in the distal tubules. This increases sodium reabsorption without altering the urine volume (111). Because the antinaturesis following aldosterone administration takes one hour to develop, Edelman et al.(29) have suggested that it acts on the enzymes involved in the transmembrane transport of sodium.

The third efferent system is the hypothalamoneurohypophyseal system. This system is controlled primarily by changes in the osmolarity of the plasma (8) although drugs or hypothalmic stimuli also release antidiuretic hormone (ADH) into the vascular system (112). The elevated plasma levels of ADH temporarily increase water reabsorption from the lumen of distal renal tubules and collecting ducts into the interstitial space (91). The ADH released by trauma retains any exogenously administered or endogenously produced water and results in a fall in the urine volume and an increase in the extracellular volume (102).

Circumventing this whole neuroendocrine reflex are a group of direct acting, biologically active stimuli which are released from traumatized cells into the plasma (110). They include histamine, serotonin and acetylcholine, and they stimulate the adrenal gland to release catecholamines. Finally, the renin reflex stimulates aldosterone release when activated by a reduction in the mean renal artery pressure (109).

In summary the following metabolic alterations occur in response to trauma. (1) Carbohydrate metabolism is affected by an increased glycogenolysis in the liver which releases glucose into the blood. (2) As glucose uptake by the peripheral cells increases, plasma lactate and pyruvate levels rise. (3) Minerals and proteins, lost from the injured or destroyed tissue, result in a negative

nitrogen balance (79). Glucocorticoids stimulate this process (116). (4) The potassium losses exceed the nitrogen losses raising the urinary potassium/nitrogen ratio from 4:1 to 12-15/1 (55). The potassium balance is restored within two to five days, or several days faster than nitrogen balance. (5) Free water clearance becomes negative, regardless of the solute load because ADH retains the water produced by the Krebs cycle or released from traumatized cells (102).

Surgical Modification of the Metabolic Response to Trauma

The metabolic response to trauma during surgery is modified by the addition of preanesthetic and anesthetic agents and by the artificial control of respiration. The peripheral and higher center stimuli which normally initiate homeostatic responses are depressed by the action of general anesthetics on the RAS and the thalamus (10). The administration of succinycholine ensures that all voluntary and involuntary higher center control of the respiratory system is lost. This leaves the quality of respiration to the clinical judgment of the attending anesthetist.

Myocardial activity continues because of the automaticity of the pacemaker (19). Under halothane anesthesia vagal activity increases as the depth of anesthesia increases, while catecholamine release is unaltered except during periods of hypoxia or acidosis (40, 21).

In addition to the metabolic changes during surgical trauma, there are several other biological factors which depend upon the site, magnitude and duration of the traumatic injury. Moore (81) lists these factors as central nervous system or airway injury, hemorrhage, edema into injured areas, hypotension, liberation of toxins into the vascular stream, loss of infection barriers and the preoperative nutritional status. To arrive at a "weighted accretion of the biological components of trauma", Moore grades all factors by their life endangering potential. In ascending order of importance they are (1) threshold stimuli (emotional stimuli, minor trauma, anesthesia, immobilization and starvation), (2) threatening challenges (hemorrhage, fluid accumulation, low flow rates and anoxia) and (3) tissue killing injuries (sepsis, necrosis and shock). Through the modalities of acidosis, anoxia, hypotension, hypovolemia or relative energy lack these biological factors modify the biochemistry of the body and stimulate the metabolic response to trauma.

Body Metabolism and Biochemistry During Extracorporeal Circulation

Extracorporeal circulation provides the respiratory and pumping actions of the heart and lungs. The first problem is to provide a sufficient flow of blood and uptake of oxygen to meet the requirements of the body during bypass.

The second problem is the technical or design problem of minimizing the trauma to the vascular system and the blood.

Clark (14) has defined the human oxygen requirements as 3.7-8.5 ml./Kg./min. or about 125-145 ml./min./m^2. (15). This can be provided by oxygen tensions of 115-150 mmHg. and flow rates of 2.2 l./min./m^2 which produce oxygen uptakes of 110-130 ml./min./m^2.(74). Hypothermic levels of 30°C reduce the basal requirement by one-third (13). The oxygen tensions are easily provided and can be maintained but the high carbon dioxide diffusion coefficient results in rapid equilibration and removal of carbon dioxide through the air vent of the pump. Therefore, carbon dioxide must be added to the oxygenating column to reduce the diffusion gradient and avoid a respiratory alkalosis.

If the carbon dioxide tension is held constant, the pH is proportional to the degree of metabolic acidosis (93). Pre and postoperative measurement of the bicarbonate concentrations, which vary directly with the oxygen consumption, are an even better indicator of any metabolic acidosis (92). When a metabolic acidosis develops, the anions which accumulate are primarily lactate, pyruvate and inorganic phosphate (69). The lactate level rises because blood and oxygen flows to the liver and to the body tissues are decreased (2). As the plasma and intracellular hydrogen ion concentrations increase, the cells lose potassium to maintain electrical neutrality (66). Hyperkalemia occurs particularly if acidosis from poor perfusion releases epinephrine which increases the glucose and

potassium lost from the liver.

Potassium Metabolism

Quantitative studies indicate that the average seventy kilogram male contains 132 grams or 3200 mEq of potassium (78). Eighty-five to ninety percent of this amount is exchangeable. Ninety-eight percent of the total body potassium is maintained within the cells while less than two percent is found in the extracellular space. The average intracellular potassium concentration lies between 130 and 160 mEq/l as calculated by isotope dilution and muscle biopsy studies (85,68). Since eighty percent of the body cell mass is muscle tissue, most of the body potassium is located within muscle cells. The average extracellular concentration is 3.6-5.4 mEq/l (7), while the average body potassium concentration is between 44 and 47 mEq/Kg. in males or 37 and 44 mEq/Kg in females (78).

Additions and losses to the potassium reservoir range from 35 to 100 mEq. per day (74). All the potassium provided by the diet is absorbed via the small bowel and is handled as follows. Losses occur into the urinary tract (90%) and the stools (10%) (45). In the kidneys, almost all of the potassium filtered by the glomeruli is reabsorbed in the proximal tubules (45). In the distal tubules potassium and hydrogen ions compete in the cation exchange process for sodium under the influence of aldosterone (111).

a. Potassium and the Cell Membrane

Using Einthoven's string galvanometer, Bernstein (9) in 1902 measured the membrane potential difference between electrodes on injured and normal cell surfaces. Nernst (89) in 1905, hypothesized that the potential difference Bernstein recorded was due to the transmembrane potassium concentration of the cell. The Donnan equilibrium equation, which was subsequently formulated to predict the potential difference across semipermeable membranes, has been found to hold true only for resting or nondepolarized membranes (16). During depolarization the potential difference is momentarily reversed and becomes proportional to the logarithm of the transmembrane sodium concentration (48). The cells lose small amounts of potassium and take up equivalent amounts of sodium (47). The strong active transport mechanism (ATP system) in the cell wall restores the depolarized cell to its original state by transporting the sodium ions out of the cell against an increasing electrochemical gradient. The negative charge created inside the cell attracts the potassium back into the cell (61).

Evidence for the ATP system is now well documented. An energy source, ATP, is present in abundance at many cell membrane sites. Its energy releasing enzyme, ATP-ase, is found in high concentrations in cells which have high transport capacities (36). Absence of an energy source

(ATP) or an oxidizable substrate (glucose or lactate),(52) or inhibition of ATP-ase (cardiac glycosides) (25), will block the entry of potassium into the cell. Raising the extracellular concentration of either potassium or sodium stimulates the entry of potassium into the cell (117). Hodgkin (46) has postulated that one potassium ion is internalized by the system for each sodium ion that is externalized. A common carrier may be involved although exceptions to this hypothesis have been found. This suggests that any ion exchange is not coupled tightly (75).

b. Potassium in the Cell

Despite the high concentration of potassium inside most body cells, the intracellular functions of potassium have not been well documented. Many enzyme reactions involving transphosphorylation, such as anaerobic glucose metabolism, are known to require potassium for activation. Potassium may be one of the catalysts which phophorylate and polymerize glucose into glycogen (32). When glycogenolysis occurs following the action of glucagon or epinephrine on the liver, potassium is lost even before the glucose efflux reaches a maximum (17). In muscle cells where glucose -6-phosphatase is not present, glucose is retained within the cell and potassium movement into the serum does not occur (31). Instead, a net potassium uptake occurs presumably from stimulation of the sodium and potassium activated ATP-ase system by the elevated serum potassium levels (117).

c. Potassium in the Serum

The serum potassium levels reflect the moment to moment net change in the absorption, excretion and movement of potassium across the cell membrane. Because the extracellular space contains only two percent of the total exchangeable potassium,minor fluctuations in the net movement of potassium across the cell membrane produce remarkable changes in the serum level (60). Therefore the ability of a change in the serum potassium level to affect the ATP-ase system is an important mechanism for buffering any serum potassium fluctuations (117). For this reason, and because sampling is simple, reliance has been placed on serum potassium level to reflect the potassium status in the body. Hammarsten et al (45) have suggested that a total body deficit of 100-200 mEq depresses the serum potassium and a deficit of 200-400 mEq or ten percent of the total body potassium depresses the serum potassium below the normal range of values. While this relationship may hold during long term alterations in the total body potassium it does not hold during acute movements of potassium across the cell membrane (82).

Hypokalemia. "The causes of potassium loss are many, the causes of hypokalemia are few."[1] The following

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[1] Moore, F.D., Metabolic Care of the Surgical Patient, Saunders, Philadelphia, 1959, p. 393.

summary of the causes of total body potassium depletion is based on a review of Holliday and Egan (54). Chronic depletion can result from either a dietary insufficiency, or prolonged bowel and urinary losses. Because potassium is present in abundance in the diet oral hypokalemia is rare, although a relative hypokalemia may develop during periods of accelerated growth and muscle formation. Chronic intestinal loss through colostomies, draining fistulae, diarrhea, prolonged gastric suctioning, or continued use of cathartics, may result in a rapid total body potassium depletion, particularly in children. Chronic urinary potassium losses occur in long standing congestive heart disease, cirrhosis, or other edema-producing diseases where high levels of aldosterone secretion exist. Diuretic administration represents the third route by which total body potassium is reduced (64). Of particular importance are the thiazide diuretics which are administered for prolonged periods of time (64).

Acute depression of the serum potassium to hypokalemic levels may occur in the chronically depleted or nondepleted body. While chronic depletion may eventually decrease the serum potassium the cause and effect relationship is not predictable. The commonest cause of acute hypokalemia is rapid cellular internalization during metabolic or respiratory alkalosis (66,104). For instance, treatment of the patient in acute diabetic acidosis with insulin and water elevates the pH, induces a polyuria and results in a rapid intracellular uptake of glucose. All

three effects remove potassium from the serum and may suddenly reduce normal or high serum levels to hypokalemic levels (1,41).

Hyperkalemia. Serum potassium concentrations exceeding 5.4 mEq/l arise most commonly from metabolic or respiratory acidosis. Retention of carbon dioxide (respiratory acidosis) from any cause raises the hydrogen ion concentration in the serum and in the cells (103). To maintain electrical neutrality inside the cell, there is an exodus of potassium into the extracellular space. In a similar fashion metabolic acidosis stimulates a compensatory intracellular potassium loss (104).

Epinephrine may produce a transient hyperkalemia by releasing liver potassium (24). Muscle trauma can have the same effect by directly releasing large quantities of intracellular potassium. Hyperkalemia can also follow an adrenalectomy or periods of acute adrenal insufficiency when a sodium diuresis occurs for lack of aldosterone and the distal tubular exhange of sodium for potassium is decreased (4). Injudicious administration of intravenous potassium (63) or rapid transfusion of stored blood (101) may lead to an elevation of the serum potassium.

Under any of these circumstances, oliguria or anuria will compound the rate and level to which the serum potassium rises, by limiting excretion of the potassium into the urine (33).

Potassium and Heart Disease

Changes in body composition vary with the stage and duration of heart disease as outlined by recent isotope dilution studies (83). In nonedematous patients, there is usually little weight change although there is a significant shift between the intracellular and extracellular spaces. The size of the intracellular space decreases. In the remaining cells there is a slight decrease in the potassium concentration and a slight increase in the sodium content. In the extracellular space the serum sodium concentration increases, while the potassium concentration decreases, and the total exchangeable sodium/potassium ratio rises from 1.0 to 1.3. All these changes are more pronounced in females.

Patients in heart failure with clinical edema characteristically have a longer duration and increased number of acute bouts of congestive heart failure. The intracellular fluid volume continues to contract while the extracellular, plasma and the red cell volumes continue to expand. The total body water remains unchanged but, because of intracellular protein and fat losses, the body weight decreases and the total body water to body weight ratio increases.

During medical treatment, the body weight, body water and body fat all decrease (84). Water loss through excretion of the extracellular fluid accounts for most of

this weight loss. Since the water loss exceeds the solute loss the serum potassium concentration returns to normal. Both the plasma and red cell volumes decrease by about ten percent with the result that the hematocrit remains unchanged. Inside the cells potassium changes are small and inconsistent. Moore (83) notes that alterations in the total exchangeable potassium are often large but vary in direction. Following successful operative treatment a brisk water diuresis occurs and gradually over the following eight to twelve months the body cell mass is restored. This confirms the observations of other investigators (90,108) who have found that changes in the total body potassium vary with the effectiveness of the medical treatment.

Potassium and Diuretics

Since the introduction of chlorthiazides in 1956 there has been an increase in the incidence of arrhythmias in digitalized patients (70). Thiazide derivatives inhibit sodium reabsorption in the proximal tubules which increases the distal tubular sodium content and the load on the sodium/potassium exchange mechanism (64). They also inhibit carbonic anhydrase and the formation of carbonic acid which reduces the hydrogen concentration in the tubular cells and increases the availability of potassium for exchange with sodium (26). Both actions increase the distal tubular secretion of potassium, which over a prolonged period may result in a

significant decrease in the total body potassium. Lockey et al.(70) have measured the total body potassium in patients on thiazides for longer than one year and have found a variable decrease to occur. Johansson et al. (56) however, have been able to detect only minor changes in the serum potassium in patients on prolonged thiazide therapy.

Potassium and Digitalis

Evidence (43,25,57) suggests that the important effect of digitalis is on the sodium and potassium activated ATP-ase system. Toxic levels of digitalis limit potassium re-entry into the cell during repolarization by inhibiting this system (53). Although Repke (97) correlated glycoside toxicity with ATP-ase inhibition, results with therapeutic glycoside levels are conflicting (36). Because the ATP-ase system is the energy source for all three phases of myocardial contraction, (excitation, excitation-contraction and contraction), it has been difficult to isolate the actions of digitalis on each phase (36). Recent studies (35) have indicated that the inotropic action of cardiac glycosides follow inhibition of the system which reaccumulates calcium ions in the vesicles lining the inside of the sarcoplasmic reticulum. Since "active" calcium ions in the sarcoplasm catalyse the contraction of actin and myosin, the duration of myofibril and myocardial contraction is prolonged (65). At the same time the sodium concentration in the sarcoplasm is elevated

for reasons which are as yet unexplained (72).

Potassium During Extracorporeal Circulation

With the increased use of extracorporeal procedures the incidence of postoperative ventricular arrhythmias has risen, particularly in procedures involving valve replacements in patients with acquired heart disease (5). Total body potassium depletion has been implicated because of the relationship of potassium to impulse conduction, and digitalis (105). Isotope dilution studies before and after surgery, have failed to reveal any marked alterations in the total body solute content or in the size of the extracellular and intracellular fluid volumes (83,84). Lockey et al.(70) have been unable to relate the amount of potassium lost in the urine during surgery to the serum potassium concentration. They have suggested that little change occurs in the body potassium balance. They also found that postbypass serum potassium levels were equal to or slightly below prebypass levels, particularly in patients on preoperative diuretic therapy. Other observers (114,6,22) have noted a more marked decrease in the serum potassium during bypass, although the evidence for this is conflicting (27). DeWall and Lillehei (20) postulated that low serum potassium levels are due to a combination of hyperglycemia and hyperventilation alkalosis. Bernard et al.(5) have modified this theory and suggested that all changes in

serum potassium are secondary to changes in the pH. They have shown that marked changes in the extracellular pH, whether metabolic or respiratory, result in predictable changes in the serum potassium.

Unfortunately the experiments performed by Barnard et al.(5) used unphysiological pH levels to prove that a direct relationship between the pH and the serum potassium did exist. The results of DeWall and Lillehei (20) are questionable since extracorporeal circulation with glucose and water as the priming solution did not produce serum potassium levels which were markedly different from cardiopulmonary procedures which used priming solutions devoid of sugar. Lockey et al.(70) did not study the serum potassium fluctuations during bypass and did not use statistical methods to analyse their results.

To determine what factors affect the serum potassium twenty-six adult cardiopulmonary operations were studied. The serum potassium pattern during surgery was documented and compared with the duration of bypass, the arterial pCO_2 pattern and the oxygen consumption pattern. Correlation coefficients have been calculated to establish the quantitative relationship between the serum potassium and the parameter under consideration. Exceptional serum potassium patterns have been examined in detail to determine whether or not the preoperative medication, the net fluid or blood balance during surgery, the total lactate addition during bypass, or the operative and postoperative urine

volumes caused the unusual pattern. Finally, results have been assessed in the light of the statistical methods used in the study, the size of the group under investigation, and the technical problems which were encountered.

CHAPTER II

METHODS AND MATERIALS

Subjects in the Study

This study is based on twenty-six consecutive adult cardiopulmonary operations, performed by the Department of Cardiovascular Surgery, in the University of Alberta Hospital. A summary of the age, sex, weight, duration of perfusion, and surgical procedure of each patient who underwent surgery is given in Table 1.

The patients ranged in age from twenty-four to seventy-two years and averaged fifty-two years of age. Seventeen patients were males and nine were females. Weights ranged from forty-eight to eighty-eight kilograms averaging sixty-six kilograms. Perfusion times ranged from twenty to one hundred and fifty-five minutes, with an average of ninety-three minutes. Repairs of congenital heart defects were the shortest procedures and multiple valve replacements were the longest. Three patients underwent atrial septal defect (ASD) closures, sixteen had single valve replacements or repairs, and four had double valve replacements. One patient had two valves replaced and a third valve repaired; another had an aortic valve and arch replaced, and the last patient had a torn chordae resutured.

All patients were placed on a diet (Appendix 1)

TABLE 1

AGE, SEX, WEIGHT, LENGTH OF BYPASS
AND SURGICAL TREATMENT

Case No.	Age	Sex	Wt. (Kg)	Length of Bypass (min.)	Surgical Treatment
1	46	M	69	98	Aortic replacement
2	42	M	83	97	Mitral replacement
3	49	F	58	155	Mitral replacement & tricuspid annuloplasty
4	55	F	61	76	Aortic replacement
5	51	F	48	98	Aortic replacement
6	57	F	50	122	Aortic replacement & mitral valvotomy
7	53	M	71	141	Mitral replacement
8	60	M	68	97	Aortic replacement
9	52	M	63	95	Aortic replacement
10	44	F	48	148	Mitral replacement & tricuspid replacement
11	52	M	72	76	Mitral replacement
12	45	M	81	112	Aortic replacement
13	42	M	88	150	Aortic replacement & tricuspid replacement
14	44	F	59	32	ASD repair
15	29	M	74	119	ASD repair & mitral plication
16	63	F	55	44	Mitral replacement
17	50	F	54	20	Mitral annuloplasty
18	64	F	50	28	ASD repair
19	51	M	70	76	Aortic replacement
20	51	M	86	92	Aortic replacement
21	68	M	69	37	Repair of torn chordae
22	43	M	82	87	Aortic replacement
23	52	M	60	99	Mitral replacement & tricuspid replacement
24	52	M	67	81	Aortic replacement
25	51	M	69	89	Aortic replacement, tricuspid annuloplasty & mitral replacement
26	44	M	86	148	Aortic replacement & mitral replacement
Means	52		66	93	

24 to 72 hours before surgery, which contained 1.3 grams (33 mEq) of potassium with each 1000 calories. This diet limited fluctuations in potassium intake, stabilized potassium excretion and limited the patient's sodium intake to 2.0 grams per day. The diet was explained to each patient once and then reviewed once during the stabilization period.

One hour prior to surgery a Levine tube and a Foley catheter were inserted and the premedication was given. Forty-five minutes before surgery the patient's blood volume was determined.

Equipment and Procedures

Blood Volume Procedures

Blood volume determinations followed a modification of the simultaneous isotope dilution method of Wood and Levitt (118). Whole blood samples were counted through windows centered on the photopeaks of Chromium 51 and Iodine 125. The Cesium-based spectrum was attenuated so that the 328 Kev chromium peak was counted with a 640 Kev base and an 8 volt window. For the iodine, a 56 Kev base and a 1 volt window setting were used for counting the 38 Kev photopeak. No iodine overlap occurred in the chromium window and chromium overlap in the iodine window never exceeded two percent of the chromium window counts. Because iodine counts were three times the chromium counts, this error was less than one half of one percent and was

considered insignificant.

All gamma emissions were counted through a sodium iodide crystal with a Nuclear Chicago Model #8725 well scintillation counter. (Appendix 2). The counting efficiency per cc. of solution was maximized by using tight fitting plastic syringes containing two millilitres of solution in a depth controlled well. Background radiation was subtracted automatically and isotope contamination of the well was checked before and after each procedure. A sample calculation sheet is given in Appendix 3.

Twenty millilitres of blood were obtained through an eighteen gauge needle and added to five millilitres of a modified Strumia solution (106) containing sixty microcuries of radioactive chromium. This mixture was incubated for fifteen minutes at room temperature for the first five determinations. The last sixteen determinations were incubated for six minutes at 37-39°C. Chromium uptake by the red cells was stopped by the addition of 250 milligrams of a reducing agent, ascorbic acid. Using plastic syringes to limit iodine adsorption, ten millilitres of this solution, containing twenty-five microcuries of Chromium-51, were injected into the patient. Using the same needle, two ml. of radioactive iodinated serum albumin (RISA), containing six microcuries of iodine-125, were injected. The remaining tagged cells were refrigerated for the postoperative determination. While the radioactive

elements mixed with the blood for twelve minutes, a RISA standard and a two ml. sample of the tagged cells were counted. Then, a post dilution two ml. sample was taken and its chromium and iodine gamma emissions were counted. Determination of the post-dilution sample hematocrit enabled the total sample counts for each isotope to be converted to counts per ml. of red cells or plasma. By dividing the respective sample counts into the total injected counts, the dilution factor or volume was isolated. The total blood volume was obtained by adding together the two volumes. After each determination, corrections were made for chromium overlap in the iodine window and for untagged chromium in the injected and sample solutions. The standard deviation of the error of this single sample method has been estimated at 10-15% (118).

The postoperative blood volume determination began one-half hour after surgery. A two millilitre sample of blood was drawn and counted before the injection of ten mls. of the originally tagged red cells and another two ml. of the RISA solution. After twenty minutes of mixing, a post equilibration sample was taken and counted. The increase in the equilibration period from twelve to twenty minutes allowed for the delay in the mixing which occurs after acute alterations in the blood volume (18). The pre-injection radioactivity levels were subtracted, thus isolating the respective dilution factors. By

comparing the pre and postoperative volumes, the net operative plasma, red cells, and total blood volume changes were identified.

Anesthetic Procedures

Premedication usually consisted of 10-15 milligrams of morphine and 0.4-0.6 milligrams of scopolamine or atropine given intramuscularly, one hour before surgery. In the operating theatre, intravenous pentobarbital, with succinylcholine as the muscle relaxant, initiated anesthesia. Ventilation from a British Oxygen Anesthetic table, provided the maintenance anesthetic of halothane, nitrous oxide and oxygen during the pre and postbypass periods. Once under anesthesia, central venous pressure and intra-arterial catheters were inserted, calibrated and attached to an Electronics for Medicine recorder Model PR-7. Also attached were a set of ECG leads for continuous monitoring during surgery.

During the bypass period, halothane (0.2%), oxygen (5 litres per bag) and carbon dioxide (0.15-.35%) were provided through an auxiliary anesthetic table to the oxygenating column of the Travenol bag as shown in Appendix 4. A vent at the top of the bag provided atmospheric equilibration and prevented a pressure build-up. The addition of carbon dioxide to the oxygen line reduced the pump-air carbon dioxide (CO_2) gradient and limited the CO_2 blowoff and the development of a respiratory alkalosis.

The second responsibility of the anaesthetist was to monitor the net fluid and blood balances and provide specific replacement therapy when necessary. To determine the net operative fluid balance, all the fluid losses were subtracted from the total fluid additions.

Fluid additions were of three types. (1) The donor blood contained 120 ml. of acid-citrate-dextrose (ACD) solution in each 500 ml. bottle which reduced the hematocrit to 35 (11). (2) The Ringer's lactate contained 4.0 mEq/l of potassium. The Travenol bag was filled with 20 ml/Kg of this solution at the start of bypass. Additional amounts were given as required during bypass . (3) The dextrose and saline kept the pressure lines open. Additions never exceeded 400 ml.

Fluid losses were also of three types. (1)Direct losses occurred into the swabs, towels, drapes and suction bottles. Swab and towel losses were calculated by subtracting the actual weight of each blood soaked swab from a predetermined dry standard weight. Drape losses were estimated, after pre and postoperative weight changes indicated that the postoperative drapes contained less than 400 ml. of fluid. (2) Urine excretion was measured directly. (3) Fluid left in the pump system after the patient was taken off the bypass system, were also measured directly.

The blood balance was determined by adding together all swab, towel, drape and suction losses, and subtracting from the total all transfused donor blood.

Replacement was usually on a ml. for ml. basis through a coil warmer using blood collected and cross matched less than 24 hours before surgery.

Bypass Procedures

Prior to bypass, patients were given heparin sulphate. All patients over 60 kilograms, or those expected to be on the pump for longer than ninety minutes, were placed on two Travenol bags filled with 20 ml/Kg of lactated Ringer's solution. Patients were then connected to the pump machine (Appendix 4), arterial and venous clamps were released and pumping started. The venous blood flowed into the Travenol bag by gravity drainage where the bloodless prime reduced the hematocrit by twenty to forty percent. After temperature modification and oxygenation the blood was pumped into the femoral artery. Retrograde filling of the aorta to the aortic valve provided whole body and coronary perfusion. In aortic valve replacements or repairs, coronary perfusion was provided separately. Flow rates usually increased for 5 - 15 minutes before stabilizing in the 30 - 50 ml /Kg range. Shortly after the onset of bypass 25 grams of Mannitol and 10 millilitres of 10% calcium gluconate were added to the pump solution. The movement of lactate into the extravascular space and urine decreased the blood level in the bag during the longer bypass procedures. Additional

lactate, up to seventy-five percent of the initial lactate load, had to be added during procedures exceeding one hour. After valve surgery, the heart or aorta were resutured, air was evacuated from the heart and aorta, the lungs were re-inflated, and aortic clamp was released and the venous drainage to the pump system was stopped. Blood remaining in the pump system was added to the femoral artery in 100 ml. increments, as long as the repaired heart maintained a CVP of less than 17-20 cm. of water. If the blood pressure could not be maintained above 70 mmHg., Isuprel (1-5 mg.) in 500 ml. of glucose and water was added intravenously. With the transition complete, any blood remaining in the pump system was measured, considered as surgical blood loss, and discarded. Protamine sulphate neutralized the circulating heparin. The chest was closed and drainage tubes from the pericardial and mediastinal spaces were externalized. All arterial and venous catheters were left indwelling for monitoring purposes, although the radial artery catheter was often removed because of clotting problems.

Sampling Procedures

Venous samples were taken one day preoperatively, ten minutes before bypass, ten minutes after bypass, ten minutes before leaving the operating room, and on the first and fifth days postoperatively. Bypass samples were taken

every fifteen to forty minutes, depending on the duration of perfusion. Arterial samples were taken with all operative venous samples. Pre and postoperative samples were collected from a peripheral vein. Pre and postbypass samples were obtained through the indwelling venous and arterial catheters and bypass samples were taken from the venous and arterial sides of the Travenol bag. Approximately $2\frac{1}{2}$ to 3 ml. of venous blood and eight ml. of arterial blood were taken at each sampling in heparin-wetted syringes.

a. Blood gas determinations. Oxygen and carbon dioxide tensions, the pH and the bicarbonate concentration were determined on all arterial samples within ten minutes of collection. Oxygen and carbon dioxide tensions were determined on the operative venous samples. All determinations were performed on a Radiometer Copenhagen Micro Astrup pH meter. The standard arterial bicarbonate concentrations were calculated from Astrup's nomogram (3). In the five hypothermic patients the results were adjusted to 38°C (103). The arterial and venous oxygen saturations were calculated from the oxygen tensions. The oxygen consumption was calculated using the Fick principle (59) on samples obtained during bypass. Calculations included oxygen combined with the hemoglobin and oxygen dissolved in the plasma. The results were converted to millilitres of oxygen consumed per minute per square meter of body surface area. A sample calculation is given in Appendix 5.

The remainder of the arterial sample was refrigerated until the end of the surgical procedure.

b. Hematocrits. After surgery, all samples were mixed for three minutes and dual microhematocrits were made using micropipettes centrifuged at 30,000 R.P.M. for 5 - 6 minutes. They were read immediately on an Adams Autocrit Centrifuge[1] and averaged for a final result. No correlations were made for trapped plasma.

c. Serum Potassium. The remainder of the arterial sample was centrifuged at 3,000 R.P.M. for one-half an hour. Two to three millilitres of plasma were removed and refrigerated until the next scheduled Technicon Auto-Analyser[2] electrolyte run.

Preoperative and first and fifth day postoperative blood specimens were collected by the University of Alberta Hospital laboratory staff. Electrolyte determinations were made on these samples using the same autoanalyser. The standard deviation of the error in this procedure is 0.1 mEq/l. (12).

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[1] Clay-Adams Inc., Springfield, Illinois.
[2] Technicon Instruments Inc., Chauncey, New York.

d. Urine. Urine formed during prebypass, bypass, post-bypass periods and the first 24 hours postoperatively was collected. Due to incomplete preoperative catheterization, catheter kinking and collection timing problems, all operative specimens were combined after the thirteenth patient. The total urine volume was measured and the potassium concentration in the urine was determined on each specimen.

Treatment of Data

Data collected on each patient has been tabled in Appendix 6. Cross-sectional and individual patient data have been analysed and discussed in Chapter III. Averages, ranged and standard deviations have been determined where indicated. Where relationships with serum potassium were thought to exist, correlation coefficients have been calculated using the Ford Dearborn computer system.[1] An example of the program used in these calculations is contained in Appendix 7. Discussions have been based on these correlations and on any exceptional cases.

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[1] Courtesy of Professor J.J. Wettlaufer, Dean, School of Business Administration, University of Western Ontario, London, Ontario.

CHAPTER III

ANALYSIS AND INTERPRETATION OF DATA

The serum potassium pattern exhibited by the twenty-six patients in the study indicated that a prebypass rise and a postbypass fall in the potassium concentration occurred. The preoperative medical therapy and the medical events which coincided with these changes have been examined. The serum potassium pattern during bypass has been examined in detail. The effects of the duration of bypass, the bypass pH, the bypass oxygen consumption and the bypass body temperature on the serum potassium concentration have been noted. Miscellaneous other factors including the blood balance, the net fluid balance, the urine formation and the arrhythmias have been examined. Finally the specific problems encountered in this study and the surgical outcome of the patients have been outlined.

The Serum Potassium Pattern During Surgery

Serum potassium values before, during and after surgery are recorded in Table II and Figure 1. From a preoperative mean of 3.9 mEq/l, the serum potassium rose 0.9 mEq/l during prebypass and dropped 0.1 mEq/l as the patient began bypass. During bypass, it rose 0.3 mEq/l to a peak of 5.0 mEq/l; then, it dropped 0.5 mEq/l after

TABLE II

SERUM POTASSIUM LEVELS BEFORE, DURING, AND AFTER CARDIOPULMONARY SURGERY.

(in mEq/l)

Case No.	Pre-Op.	Pre Bypass	Early Bypass	Late Bypass	Post Bypass		Post Operative Day 1	Post Operative Day 5
1	4.0	4.2	4.7	4.7	4.7	3.5	5.0	4.2
2	3.4	4.8	4.1	4.7	4.3	4.8	4.9	4.0
3	3.1	4.5	3.7	4.9	4.8	4.7	3.5	3.8
4	3.9	5.3	3.5	4.5	4.9	4.7	3.3	3.7
5	4.1	4.4	4.0	4.1	3.9	4.3	3.4	3.1
6	3.8	5.2	4.6	5.7	4.1	3.8	died day #1	
7	3.9	6.0	5.8	4.3	3.9	4.7	6.5	died day #2
8	4.6	5.7	5.7	6.0	5.7	4.7	3.9	4.2
9	3.2	4.2	4.1	4.5	3.3	2.9	4.0	3.8
10	4.1	5.0	4.6	5.0	4.9	-	4.0	4.0
11	3.3	4.3	5.6	6.3	4.6	4.2	4.1	2.8
12	3.6	5.0	4.9	5.4	4.2	3.6	4.6	3.2
13	4.2	6.2	5.7	5.5	5.1	-	4.2	4.1
14	3.8	4.7	4.5	4.7	4.3	-	4.1	N.A.*
15	4.6	4.4	4.7	4.8	4.3	4.0	4.1	3.8
16	4.2	4.4	5.0	5.0	4.5	-	4.9	N.A.
17	4.1	4.3	4.4	4.4	4.1	3.7	4.8	3.9
18	4.1	5.0	4.7	4.9	4.0	4.3	5.1	3.8
19	4.1	5.7	5.5	5.6	5.0	4.9	4.8	3.9
20	4.4	4.9	4.9	5.2	4.6	4.6	4.0	3.6
21	4.0	5.0	5.4	6.1	6.0	4.8	4.2	3.5
22	3.8	4.8	4.8	5.0	4.5	-	4.2	4.0
23	3.5	4.5	4.7	4.5	3.7	3.6	4.1	4.3
24	3.9	4.6	4.9	5.7	4.7	-	4.5	3.2
25	4.7	4.5	3.9	4.5	4.3	3.8	4.0	4.3
26	3.7	4.1	4.4	4.5	5.8	5.4	3.7	3.6
Means	3.94	4.82	4.72	5.02	4.54	4.26	4.73	3.77
Range of Values	1.5	2.1	2.3	2.2	2.7	1.9	1.8**	1.5

* N.A. - not available; patient discharged

** Case seven is excluded

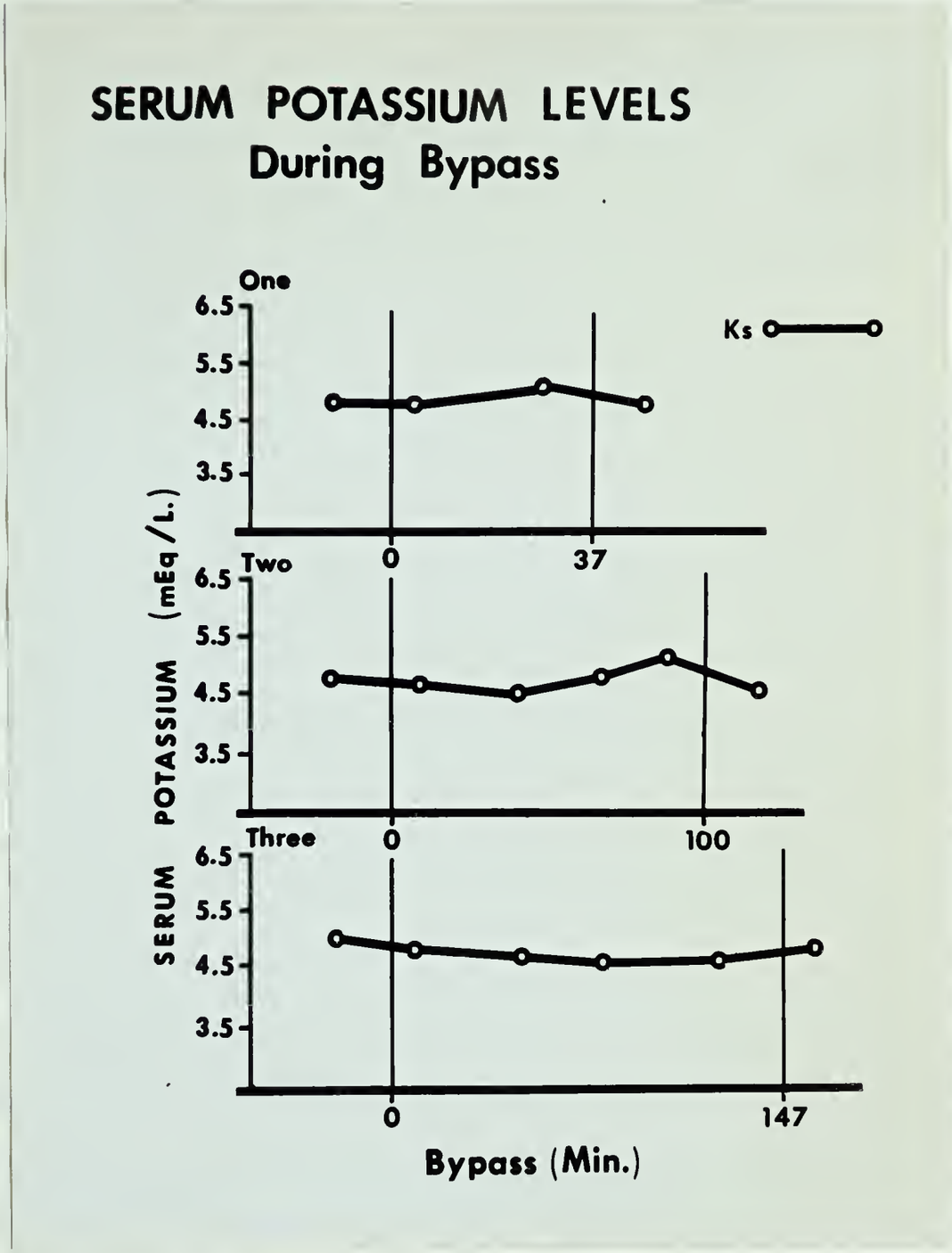

SERUM POTASSIUM LEVELS
During Bypass
One
Two
Three
Ks
6.5
5.5
4.5
3.5
SERUM POTASSIUM (mEq/L.)
0
37
100
147
Bypass (Min.)

Figure 1

bypass, rose to 4.7 mEq/l on day one and then dropped to 3.8 mEq/l by the fifth postoperative day. The high and low values differed by 1.5 mEq/l preoperatively, by 2.7 mEq/l after bypass, and by 1.5 mEq/l on the fifth post-operative day.

The operative pattern has been divided into three sections (Figure 1).

The Ascending Limb

The mean arterial blood pressure dropped markedly during the prebypass period from the action of halothane on the parasympathetic system. Atropine was administered if the systolic pressure dropped below 80 mmHg. During the prebypass period the urine formation averaged twenty-two ml. and the urine potassium content averaged 2.5 mEq/l in those patients in whom it was obtained. (Table III). In the four patients who were hypotensive before bypass (Table IV) with systolic blood pressures of less than 75 mmHg., the serum potassium rose an average of 1.6 mEq/l. The average rise in the remaining patients was 0.8 mEq/l. The probability of all four cases exhibiting such a rise is significant at the $p = 0.0001$ level (49,50). Concurrent with the rapid rise in the serum potassium was a reduction in the urine volume and the amount of potassium excreted in the urine (Table III).

The origin of the serum potassium elevation could

TABLE III

URINE FORMATION DURING SURGERY

Case No.	Urine Potassium (mEq)				Urine Volume (cc)			
	Bypass			Total	Bypass			Total
	Pre	During	Post		Pre	During	Post	
1	3.9	2.7	9.0	15.6	50	42	180	272
2	5.0	2.7	2.5	10.2	40	50	40	130
3	4.1	3.1	2.8	10.0	53	50	38	141
4	0.0	4.5	1.2	5.7	0	80	40	120
5	2.5	5.9	2.8	11.2	20	95	40	155
6	2.1	3.4	0.8	6.3	45	130	28	213
7	No Foley			13.0	-	-	-	300
8	8.0	6.6	8.2	22.8	45	90	130	265
9	2.3	8.2	13.4	23.9	40	200	210	450
10	0.7	9.8	1.0	11.5	11	170	22	203
11	0.8	6.0	4.0	10.8	20	90	110	220
12	No Foley			16.9	-	-	-	300
13	2.7	13.6	5.5	21.8	25	140	65	230
14	No Foley			15.8	-	-	-	195
15	1.8	8.3	6.9	17.0	15	170	150	335
16	0.8	4.9	0.8	6.5	20	80	20	120
17	0.0	8.7	5.2	13.7	0	160	100	260
18				4.1	20	15	25	60
19				24.0	25	145	130	300
20				8.0	0	80	40	120
21				10.9	30	10	170	210
22				16.6	20	210	185	415
23				10.4	15	110	55	180
24				4.4	20	40	10	70
25				18.3	20	130	70	330
26				9.0	0	150	85	235
Means	(2.5)	(6.3)	(4.6)	12.2	22	101	81	204

TABLE IV

MEDICAL TREATMENT AND SURGICAL COMPLICATIONS

Case No.	Preoperative Therapy	Surgical Complications	Postoperative Complications/ Therapy
1			
2	Digitalis		Atrial Fibrillation Redigitalized
3	Digitalis		Redigitalized
4	Digitalis Diuretics		Redigitalized
5	Digitalis Diuretics		Redigitalized
6		Prebypass Hypotension, Atrial Tachycardia & Atrial Fibrillation during Bypass.	Died day one
7		Prebypass Hypotension, Hypothermic Bypass, Ventricular Fibrillation during Bypass.	Redigitalized Died day two
8	Diabetic on Tolbutamide		Digitalis
9	Digitalis Diuretics		Atrial Fibrillation continued Redigitalized
10	Digitalis	Hypothermic Bypass. Atrial Fibrillation during Bypass, Spontaneous Defibrillation.	
11	Digitalis Diuretics Potassium Chloride		
12		Hypothermic Bypass, Ventricular Fibrillation during Bypass.	
13		Prebypass Hypotension, Isuprel required post-bypass.	Digitalis
14			
15		Hypothermic Bypass, Atrial Fibrillation during Bypass, Spontaneous Defibrillation.	Digitalis

TABLE IV cont'd.

Case No.	Preoperative Therapy	Surgical Complications	Postoperative Complications/ Therapy
16	Digitalis	Atrial Fibrillation during Bypass	Redigitalized
17			
18			Arrested day two Resuscitated Digitalis for Bigeminal rhythm
19			Digitalis
20	Diuretics		Digitalis
21		Prebypass Hypotension	Digitalis
22	Digitalis		Postoperative Extrasystoles Redigitalized
23	Digitalis Diuretics		Redigitalized
24			
25		Hypothermic Bypass, Atrial Fibrillation during Bypass, Spontaneous Defibrillation.	Digitalized Reoperation one month later. Died Postoperatively.
26	Digitalis Diuretics	Atrial and Ventricular Fibrillation	

have been from a reduction in potassium excretion, from potassium released by traumatized cells, from potassium added in the transfused blood, or from acidosis during the period of inadequate perfusion. The pH however was alkaline and the potassium concentration in the transfused donor blood ranged from 3.5 - 6.0 mEq/l. Potassium could have been released from red cell or muscle fiber trauma but since trauma occurred throughout the whole operation it is unlikely that there was a sudden release of potassium which could not be incorporated into other viable cells. This leaves the reduction in the urine volume as the most probable cause of the serum potassium elevation in patients with an uncorrected hypotension during the prebypass period.

The Bypass Plateau

See "The Serum Potassium Pattern During Bypass" for a detailed analysis.

The Descending Limb

After bypass the serum potassium concentration dropped an average of 0.7 mEq/l. At the same time the formation of urine recommenced, the systemic pressure rose abruptly and the hematocrit increased sharply to prebypass levels. These changes suggested that the Ringer's lactate still present in the circulatory system was passing into the extracellular space and/or the urine. Correlation coefficients (Table V)(51) indicate that the volume of

TABLE V

CORRELATION COEFFICIENTS (51)

	'r'
1. Bypass Serum Potassium (actual values) - Duration of Perfusion	
a) All Cases	.16
b) Less case seven	.20
2. Bypass Serum Potassium (first value = zero) - Duration of Perfusion	
a) All cases	.19
b) Less case seven	.38
c) Less cases six, seven, thirteen and twenty-one	.41
d) Less cases one, six, seven, twelve, thirteen, fifteen and twenty-one	.49
3. Bypass Serum Potassium - Bypass pH (first value = zero in both)	
a) All Cases	.33
b) Less cases six, seven, thirteen and twenty-one	.36
c) Less cases one, six, seven, twelve, thirteen, fifteen and twenty-one	.33
4. Bypass Serum Potassium - Bypass Oxygen Consumption (first values = zero in both)	
a) All nineteen cases	.13
b) Less cases thirteen and twenty-one	.11
5. Bypass Serum Potassium - Bypass pCO_2 (first values = zero in both)	
a) All Cases	-.17
b) Less cases six, seven, thirteen and twenty-one	-.11
c) Less cases one, six, seven, twelve, thirteen, fifteen and twenty-one	-.34
6. Operative Urine Volume (cc.) and the:	
a) Total Lactate (cc.)	.51
b) Total Fluid Gain (cc.)	.32
c) Urinary Potassium Content (mEq.)	.77
7. Urine Volume (cc./Kg.) and:	
a) Total Lactate (cc/Kg.)	.38
b) Total Fluid Gain (cc/Kg.)	.18
c) Urinary Potassium Content (mEq.)	.80
8. Net Lactate and Saline Gain (cc.) and the:	
a) Urine Volume (cc.)	.53
b) Urinary Potassium Content (mEq.)	.52
c) Duration of Perfusion	.62
9. Net Fluid Gain (cc.) - Urinary Potassium (mEq.)	.22

Ringer's lactate added to the pump system during bypass was a factor which contributed to the amount of potassium excreted in the urine. The correlation coefficient between the amount of Ringer's lactate added during surgery and urine volume was 0.51. The urine volume and the potassium content had a correlation coefficient of 0.77. On this basis it was concluded that the postbypass drop in the serum potassium was affected by the amount of lactate added during surgery. Closer examination of this relationship could be made if the urine volume and the potassium content of the urine were measured using catheters in the ureters with samples taken at intervals of every one to five minutes.

Five serum potassium patterns were unusual. Case seven, who died twenty-four hours postoperatively in acute renal failure, had a rising BUN, a falling urine volume and a serum potassium of 6.5 mEq/l twelve hours after surgery. Prolonged periods of hypotension occurred before bypass, after bypass and after surgery. Uncontrolled bleeding from the posterior myocardium precipitated a second operation immediately after the first. It was partially successful in controlling the bleeding but the systemic pressure never reached 100 mmHg. The sensitivity of the serum potassium to hypotension, oliguria and anuria was well indicated in this case.

Case nine had serum potassium levels of 3.3 and 2.9 mEq/l after bypass, following a late bypass level of

4.5 mEq/l. This 1.6 mEq/l drop to hypokalemic levels after bypass may have been due to the excretion of 13.4 mEq. of potassium in 210 ml. of urine after bypass. These figures were the largest of those recorded and support the Ringer's lactate load-urine potassium relationship suggested in the discussion of the postbypass serum potassium drop.

Case eleven was the only patient to have a pre and postoperative potassium level below 3.6 mEq/l. He was also the only patient to have potassium chloride preoperatively and was one of the five patients on digitalis and diuretics. During bypass, his serum reached a peak level of 6.3 mEq/l which was 3.0 mEq/l higher than the preoperative level. The reason for this liability was not evident.

Case twenty-one also had a marked rise in the serum potassium to a peak of 6.1 mEq/l by the end of bypass which was 2.1 mEq/l higher than the preoperative level. This elderly patient was hypotensive during the prebypass period and formed only 10 ml. of urine during his 37 minutes of extracorporeal circulation. No arrhythmic patterns developed in either this case or in case eleven during the hyperkalemic periods.

Case twenty-six exhibited an acute rise of 1.3 mEq/l to 5.8 mEq/l immediately after bypass. Concurrently, the arterial pH dropped from 7.43 to 7.32 units, the arterial pCO_2 rose from 33.5 to 45 mmHg., and the venous pCO_2 rose from 45 to 61 mmHg. Inadequate perfusion

immediately after bypass, was most likely the cause of the respiratory acidosis and the potassium rise. The prolonged pump time (148 minutes) and the extensiveness of the surgery, (annuloplasty plus double valve replacement), may have contributed toward the development of the acidosis (34).

The Effects of Preoperative Digitalization

Twelve patients received digitalis preoperatively (Table IV). When digitalized and nondigitalized patients were compared (Table VI), the mean preoperative and operative serum potassium levels in the former group were 0.1 to 0.8 mEq/l lower than the latter. The mean serum potassium concentrations were 0.1 mEq/l in reverse on the fifth postoperative day indicating that any preoperative digitalis effect was lost during surgery.

Three of the twelve digitalized patients developed arrhythmias during surgery while five occurred in the fourteen nondigitalized patients.

The Effects of Preoperative Diuretic Therapy

Seven patients received diuretic therapy preoperatively (Table IV). When diuretic and nondiuretic patients were compared (Table VII), the mean preoperative and operative serum potassium levels in the diuretic patients were 0.2 to 0.7 mEq/l lower than in the nondiuretic patients. These differences vanished by the fifth postoperative day indicating that any preoperative diuretic effect was lost

TABLE VI

THE EFFECT OF PREOPERATIVE DIGITALIZATION ON SERUM POTASSIUM LEVELS (in mEq./l.)

Group	Number in Group	Pre Operative	Bypass				Post Operative	
			Pre	Early	Late	Post	Day 1	Day 5
A	12	3.7	4.5	4.3	4.3	4.4	4.0	3.9
B	14	4.1	5.0	4.9	5.1	4.5	4.5	3.8

Group A -- Patients on digitalis therapy preoperatively.

Group B -- Patients not on digitalis therapy preoperatively.

TABLE VII

THE EFFECT OF PREOPERATIVE DIURETIC THERAPY ON SERUM POTASSIUM LEVELS (in mEq./l.)

Group	Number in Group	Pre Operative	Bypass				Post Operative	
			Pre	Early	Late	Post	Day 1	Day 5
A	7	3.8	4.6	4.4	4.7	4.3	3.9	3.7
B	19	4.0	4.9	5.1	5.1	4.6	4.4	3.9

Group A -- Patients on diuretic therapy preoperatively.

Group B -- Patients non on diuretic therapy preoperatively.

during surgery.

Two of the seven patients developed operative arrhythmias, as did six of the nineteen patients not on diuretics.

The Effects of Preoperative Digitalis and Diuretic Therapy

Five patients received digitalis and diuretic therapy preoperatively (Table VIII). When those patients on both drugs were compared with those not on both drugs, the former were 0.3 to 0.8 mEq/l lower than the latter. The two groups had identical mean serum potassium concentrations by the fifth postoperative day.

Only one patient developed an arrhythmia during surgery.

The trend in all three groups was for the differences in the mean serum potassium concentration to increase during surgery and bypass and to narrow postoperatively. At no time were the differences between the groups under consideration significant at the $p = 0.1$ level. The fact that therapeutic versus non-therapeutic groups were not significantly different poses some interesting questions. If all the patients were adequately digitalized and/or took all their diuretic medication as prescribed, then it would appear that the serum potassium was unaffected. This is important since it is likely that the patients on preoperative therapy were in a later stage of failure and

TABLE VIII

THE EFFECT OF PREOPERATIVE DIGITALIS AND DIURETIC THERAPY ON SERUM AND POTASSIUM LEVELS (in mEq./l.)

Group	Number in Group	Pre Operative	Bypass				Post Operative	
			Pre	Early	Late	Post	Day 1	Day 5
A	5	3.7	4.5	4.1	4.4	4.8	3.7	3.7
B	21	4.0	4.9	4.9	5.2	4.6	4.5	3.8

Group A -- Patients on digitalis and diuretic therapy preoperatively.

Group B -- Patients non on digitalis and diuretic therapy preoperatively.

therefore would have had a greater reduction in their body cell mass and intracellular potassium concentration. Because longitudinal measurement of the changes in the extracellular and intracellular spaces and the adequacy of preoperative digitalization were not made, it is impossible to state whether each patient underwent an adequate therapeutic trial. The trend toward lower preoperative levels in the patients on medication could have been the result of inadequate therapy with subclinical congestive heart failure, extracellular fluid retention and dilution of the serum potassium concentration (83).

The Serum Potassium Pattern During Bypass

Three groups were formed (Table IX) for the analysis of the serum potassium pattern during bypass. In Group I there were five cases who were on bypass less than one hour, four of whom were congenital heart defects. In Group II there were sixteen patients who were on bypass between one and two hours. Most of them were single valve replacements. In Group III there were five patients who were on bypass longer than two hours. They included multiple valve replacements or complicated single valve replacements.

The Effects of the Duration of Perfusion

The mean bypass values in each group are listed

TABLE IX

GROUPING BY DURATION OF PERFUSION

Group	Number in Group	Range of Perfusion Time	Average of Perfusion Times	Operative Diagnosis and Treatment
I	5	less than 1 hour	35 min.	Congenital heart diseases
II	16	1 - 2 hours	100 min.	Single valve replacements
III	5	greater than 2 hours	147 min.	Multiple valve replacements or Single valve replacements with complications

in Table X and plotted in Figure 2. The mean bypass potassium level rose from 4.8 to 5.0 mEq/l in Group I and from 4.6 to 5.1 mEq/l in Group II. In Group III, the rise occurs only if case seven is excluded.

The serum potassium suddenly decreased more than 0.5 mEq/l between two consecutive samples in cases one, twelve and fifteen. (Table XI), In case one, the pH dropped 0.1 units which indicated that a relative acidosis had developed. In case twelve, no reason could be found for the drop. In case fifteen, the pH suddenly dropped 0.15 units and the serum potassium rose to maintain electrical neutrality. In case seven, the progressive drop during bypass may reflect excretion of the potassium which had accumulated during the prebypass hypotension.

When the actual serum potassium values were correlated with the duration of bypass, the 'r' value equaled 0.16 (Table V). When each patient was used as his own control the 'r' values rose to 0.19. By excluding case seven, it rose to 0.38 and by also excluding the four patients with prebypass hypotension and Cases 1,12 and 15, it rose to 0.49 (Table V). On this basis, it is concluded that the duration of perfusion was one of the factors which determined the magnitude and direction of the serum potassium changes during perfusion.

Since the serum potassium reflects the net movement of potassium across cell membranes and into the urine,

TABLE X

MEAN SERUM POTASSIUM LEVELS DURING BYPASS

(in mEq/l.)

Group	Number in Group	Bypass					
		Pre	1	2	3	4	Post
I	5	4.7	4.8	5.0	-	-	4.6
II	16	4.8	4.6	4.6	4.9	5.1	4.4
III	5	5.1	4.8	4.8	4.7	4.7	4.8
Weighted Means		4.8	4.7	-	-	-	4.5

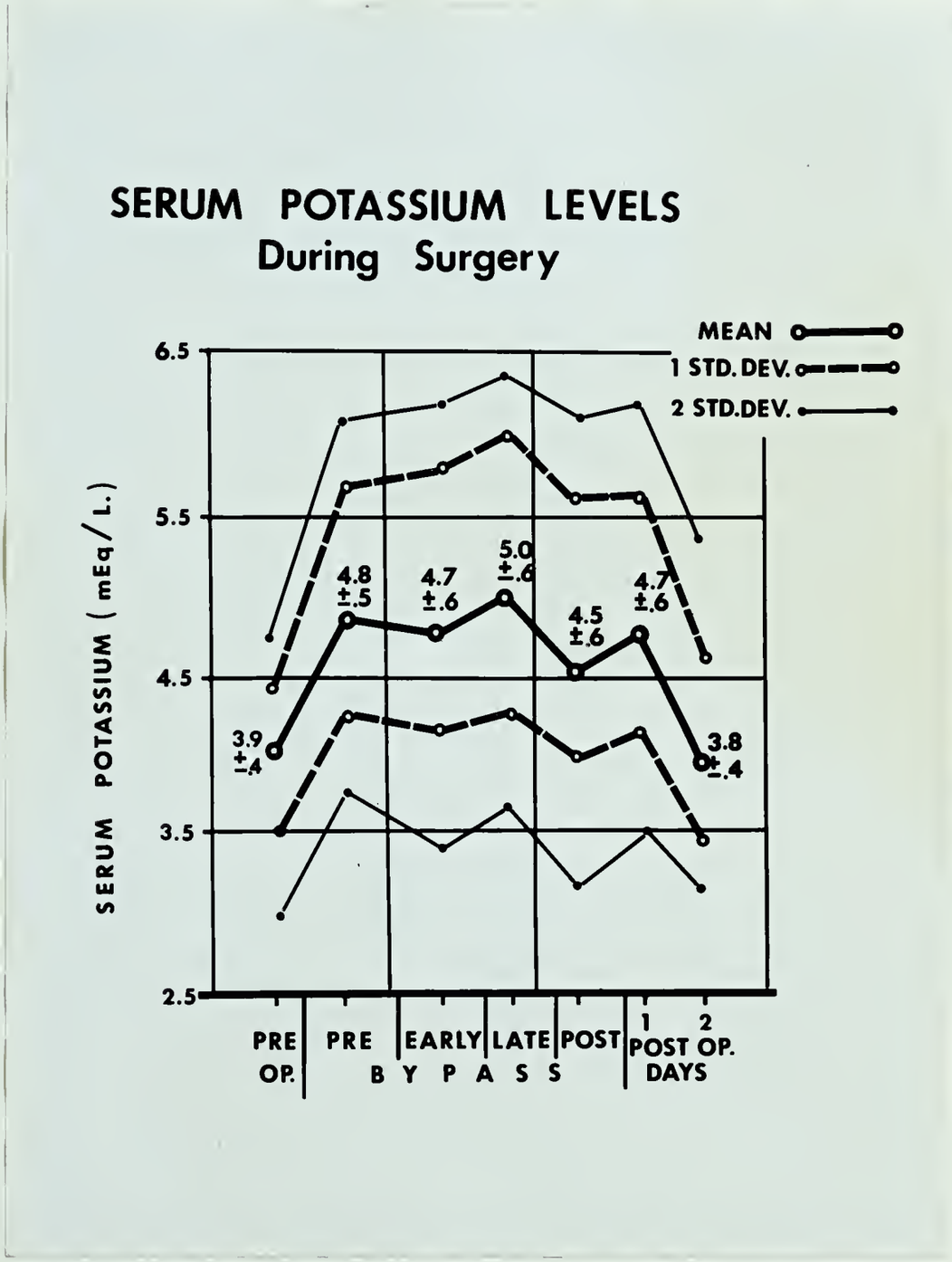

SERUM POTASSIUM LEVELS
During Surgery
MEAN
1 STD. DEV.
2 STD.DEV.
SERUM POTASSIUM (mEq / L.)
6.5
5.5
4.5
3.5
2.5
3.9 ±.4
4.8 ±.5
4.7 ±.6
5.0 ±.6
4.5 ±.6
4.7 ±.6
3.8 ±.4
PRE OP.
PRE BY PASS
EARLY
LATE
POST
1 2 POST OP. DAYS

Figure 2

TABLE XI

SERUM POTASSIUM LEVELS DURING BYPASS

(in mEq./l.)

Case No.	Group	Bypass					
		Pre	1	2	3	4	Post
1	II	4.2	4.7	3.9	4.5	4.7	4.7
2	II	4.8	4.1	4.3	4.4	4.7	4.3
3	III	4.5	3.7	4.0	4.3	4.9	4.8
4	II	5.3	3.5	3.7	4.3	4.5	4.9
5	II	4.4	4.0	3.8	3.8	4.1	3.9
6	II	5.2	4.6	4.6	5.6	5.7	4.1
7	III	6.0	5.8	5.5	4.7	4.3	3.9
8	II	5.7	5.7	5.5	5.5	6.0	5.7
9	II	4.2	4.1	4.3	4.4	4.5	3.3
10	III	5.0	4.6	4.6	4.6	5.0	4.9
11	II	4.3	5.6	5.5	5.8	6.3	4.6
12	II	5.0	4.9	4.3	5.2	5.4	4.2
13	III	6.2	5.7	5.5	5.6	5.5	5.1
14	I	4.7	4.5	4.7	-	-	4.3
15	II	4.4	4.7	4.1	4.3	4.8	4.3
16	I	4.4	5.0	5.0	-	-	4.5
17	I	4.3	4.4	4.4	-	-	4.1
18	I	5.0	4.7	4.9	-	-	4.0
19	II	5.7	5.5	5.1	5.5	5.6	5.0
20	II	4.9	4.9	5.0	5.0	5.2	4.6
21	I	5.0	5.4	6.1	-	-	6.0
22	II	4.8	4.8	4.7	4.7	5.0	4.5
23	II	4.5	4.7	4.8	4.6	4.5	3.7
24	II	4.6	4.9	5.1	5.3	5.7	4.7
25	II	4.5	3.9	4.5	4.5	4.3	3.8
26	III	4.1	4.4	4.5	4.4	4.5	5.8

then either the urinary excretion must have decreased, or the cells must have lost potassium. Insufficient data is available to determine which factor was dominant but the urine volume did decrease during bypass as the mannitol effect diminished. Not known either was the effect of trauma from bubble oxygenation on the release of potassium from red cells.

The Effects of the Bypass Bicarbonate and Hydrogen Ion Concentrations, and the Carbon Dioxide Tensions

All arterial bicarbonate concentrations were converted to standard bicarbonate concentrations (3). The mean standard bicarbonate levels during bypass remained within a narrow range of 23.9 - 19.7 mEq/l (Table XII), or just below the normal range of 24.5 - 21.3 mEq/l. The means dropped slightly with the onset of bypass, recovered during bypass and changed minimally after bypass. In Groups II and III a slight drop secondary to the reduced carbon dioxide tension (pCO_2) occurred in interval four. The minor bicarbonate depression could have occurred for one of two reasons. Either too much bicarbonate was neutralized by an excess of acid (metabolic acidosis) or carbon dioxide was lost faster than it was formed (respiratory alkalosis). By plotting all the bypass pCO_2 - pH values on an acid-base nomogram (30), (Figure 3) it is evident that the primary factor which controlled the pH was the rate at which carbon dioxide was lost.

TABLE XII

MEAN STANDARD SERUM BICARBONATE CONCENTRATIONS DURING BYPASS

(in mEq./l.)

Group	Number in Group	Bypass					
		Pre	1	2	3	4	Post
I	5	22.6	19.9	20.4			21.6
II	16	24.0	21.6	23.1	23.9	22.6	22.5
III	5	22.4	20.9	20.8	22.1	19.7	21.3
Weighted Means		23.4	21.1	-	-	-	22.1

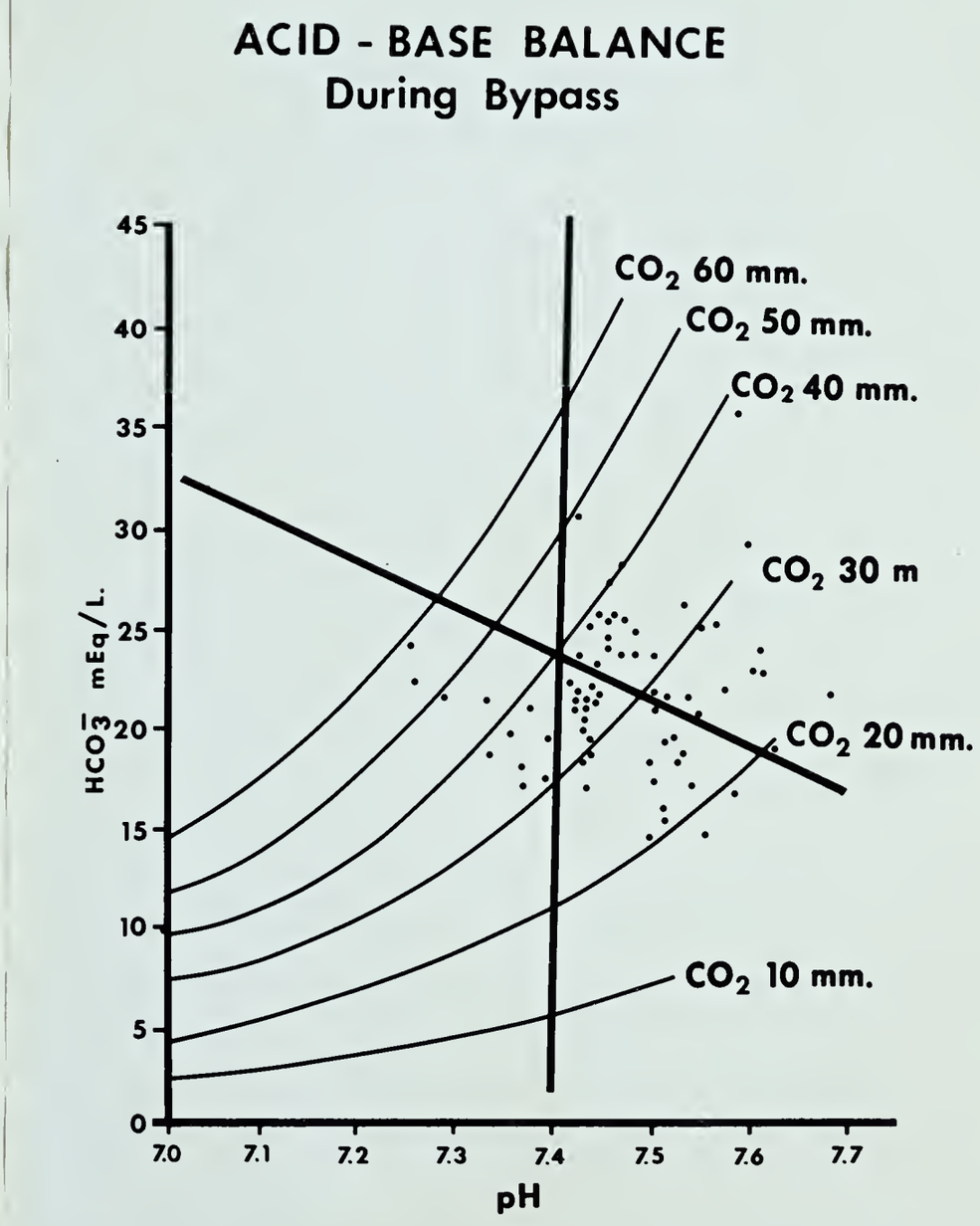

ACID - BASE BALANCE
During Bypass
45
40
35
30
25
20
15
10
5
0
HCO3 mEq/L.
7.0
7.1
7.2
7.3
7.4
7.5
7.6
7.7
pH
CO2 60 mm.
CO2 50 mm.
CO2 40 mm.
CO2 30 m
CO2 20 mm.
CO2 10 mm.

Figure 3

In all three groups the mean bypass pCO_2 dropped below the normal range, particularly in the fourth intervals of Groups II and III (Table XIII). Several exceptional patterns were noteworthy.(Table XIV). In case eighteen, the prebypass arterial pCO_2 of 38 mmHg., rose to 58 and 48 mmHg on bypass. High carbon dioxide levels in the oxygen line elevated the bypass arterial pCO_2 and limited the venous-arterial pCO_2 drop across the oxygenating column to 0 and 5 mmHg. For the same reason, the case nineteen arterial pCO_2 changed from 26 to 50 and 26 mmHg in intervals one, two and three while the venous pCO_2 changed from 35 to 48 and 41 mmHg. In both cases, the serum potassium and the serum pH dropped. In case eleven, the interval four venous-carterial pCO_2 difference suddenly increased thirteen mmHg because the CO_2 was prematurely discontinued. Despite the 0.21 unit rise in the pH to 7.67, the serum potassium rose 0.5 mE /l. In all three cases the hydrogen ion and the serum potassium levels moved in opposite directions throwing doubt on whether the direct potassium-hydrogen ion relationship exists during cardiopulmonary surgery (78).

The mean pH's which ranged from 7.35 to 7.53 units, (Table XV) dropped when bypass commenced, rose slowly during bypass and then dropped to their lowest levels after bypass. Group I followed this pattern closely. In Group II the drop onto bypass did not occur, and in Group III there was no directional change during bypass and

TABLE XIII

MEAN ARTERIAL CARBON DIOXIDE TENSIONS DURING BYPASS

(in mmHg.)

Group	Number in Group	Bypass					
		Pre	1	2	3	4	Post
I	5	33.1	33.7	33.0	-	-	39.3
II	16	35.6	34.8	33.9	33.3	26.0	37.5
III	5	38.0	30.0	27.5	32.1	28.1	36.4
Weighted Means		35.5	33.5	32.2	-	-	37.6

TABLE XIV

ARTERIAL CARBON DIOXIDE TENSIONS (mmHg) DURING BYPASS

(Temperature corrected if below 35° C)

Case No.	Group	Bypass					
		Pre	1	2	3	4	Post
1	II	32	36	39	37	25.5	31.5
2	II	43	37.5	32	35	34	33.5
3	III	57	36.5	36.5	40	37	30.5
4	II	28	33.5	36	37	30	25
5	II	27	39	35.5	43	33.5	38
6	II	43	37	30.5	25.5	27.5	32.5
7	III	46	33	27	29	29	38
8	II	40	24	24	32	22	38
9	II	40	34	29	31	31.5	45.5
10	III	26	38.5	30.5	32.5	31	35
11	II	31	32.5	39	40	21	34
12	II	32.5	37.5	36	38	23.5	32.5
13	III	34.5	23	24.5	18	22	33.5
14	I	26	24.5	34.5	-	-	51
15	II	34	55	31.5	38	30	43
16	I	31	34.5	38	-	-	34
17	I	34.5	30	25	-	-	32.5
18	I	38	58	48	-	-	49
19	II	44	25.5	50	25.5	19	35.5
20	II	33.5	29	28.5	27	23.5	39
21	I	36	21.5	19.5	-	-	30
22	II	32.5	38.5	33.5	31	29	42
23	II	39	37	31.5	38	26	50
24	II	31	33	35	24	21	38
25	II	34	33.5	34	25	22	44
26	III	26.5	28.5	22	39	33.5	45

TABLE XV

THE MEAN ARTERIAL pH DURING BYPASS

Group	Number in Group	Bypass					
		Pre	1	2	3	4	Post
I	5	7.48	7.39	7.44	-	-	7.37
II	16	7.43	7.43	7.45	7.47	7.53	7.40
III	5	7.46	7.44	7.49	7.46	7.45	7.43
Weighted Means		7.45	7.43	-	-	-	7.40

only a two unit drop in coming off bypass. In Case 26 (Table XVI) the pH rose 0.21 units in coming onto bypass but dropped 0.19 units between intervals two and three, when the anesthetist became aware of the low carbon dioxide level in the rebreathe system and increased it. Cases 11, 18 and 19 have been discussed previously. In Cases 3 and 4, the pH rose 12 and 8 units when going off bypass because of an uncompensated loss of carbon dioxide. In contrast, Cases 19, 20 and 23 had marked pH drops of 25, 28 and 33 units, due primarily to postbypass elevations in the arterial pCO_2 of 11, 16 and 24 mmHg. from subnormal late bypass levels of 22, 23 and 26 mmHg. The relationship between the mean pH in each group and the mean serum potassium level is shown in Figure 4. The coefficient of correlation was 0.33 (Table V), which contradicts the well documented inverse relationship between them (78). Factors with greater influence than the pH must control the serum potassium fluctuations during bypass.

The Effects of the Oxygen Consumption on the Serum Potassium

The oxygen consumption was determined by the Fick Method (59) in 19 of the 26 cases. The individual results (Table XVII) ranged from 28 to 106 ml./min./m^2 of body surface area. In Groups I and II (Figure 5) the mean oxygen consumption rose eight and six units during perfusion. In Group III a rise would have occurred if the exceptional

TABLE XVI

ARTERIAL pH LEVELS DURING BYPASS

(TEMPERATURE CORRECTED IF BELOW 35°C)

Case No.	Group	Bypass					
		Pre	1	2	3	4	Post
1	II	7.55	7.45	7.42	7.47	7.52	7.45
2	II	7.42	7.41	7.50	7.47	7.48	7.40
3	III	7.38	7.43	7.45	7.44	7.46	7.52
4	II	7.51	7.44	7.42	7.46	7.49	7.57
5	II	7.45	7.33	7.36	7.33	7.37	7.40
6	II	7.31	7.36	7.42	7.49	7.44	7.38
7	III	7.59	7.48	7.52	7.53	7.51	7.55
8	II	7.40	7.51	7.52	7.44	7.51	7.34
9	II	7.42	7.52	7.55	7.56	7.59	7.48
10	III	7.51	7.35	7.39	7.42	7.44	7.35
11	II	7.40	7.47	7.44	7.45	7.67	7.44
12	II	7.47	7.41	7.43	7.45	7.61	7.46
13	III	7.45	7.51	7.49	7.55	7.49	7.40
14	I	7.53	7.44	7.41	-	-	7.28
15	II	7.40	7.25	7.37	7.34	7.43	7.27
16	I	7.36	7.30	7.42	-	-	7.38
17	I	7.41	7.43	7.51	-	-	7.44
18	I	7.46	7.25	7.28	-	-	7.30
19	II	7.50	7.60	7.41	7.51	7.62	7.37
20	II	7.44	7.43	7.49	7.52	7.60	7.32
21	I	7.41	7.53	7.58			7.43
22	II	7.44	7.37	7.42	7.47	7.49	7.37
23	II	7.38	7.42	7.53	7.57	7.64	7.31
24	II	7.45	7.42	7.44	7.56	7.56	7.43
25	II	7.37	7.39	7.43	7.57	7.50	7.38
26	III	7.39	7.50	7.62	7.43	7.43	7.32

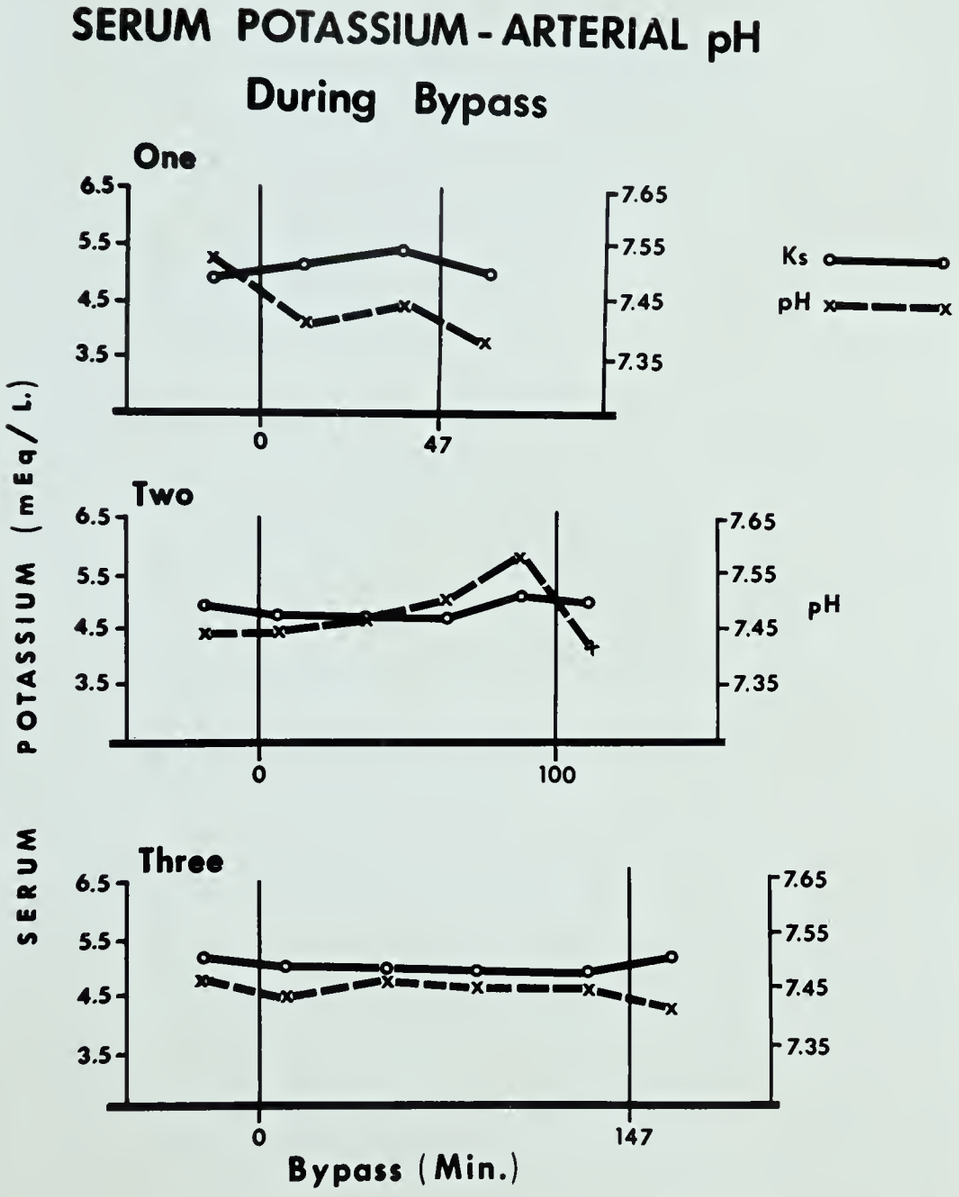

SERUM POTASSIUM - ARTERIAL pH
During Bypass
One
Two
Three
6.5
5.5
4.5
3.5
7.65
7.55
7.45
7.35
0
47
100
147
Ks
pH
SERUM POTASSIUM (mEq/L.)
Bypass (Min.)

Figure 4

TABLE XVII

MEAN OXYGEN CONSUMPTION DURING BYPASS

(ml./min./m^2)

Group	Number in Group	Mean Surface Area (m^2)	Bypass 1	2	3	4
I	5	1.64	48	63	-	-
II	11	1.83	62	66	66	72
III	3	1.92	72	63	62	73
Weighted Means		1.79	61	64	-	-
Range			75	48	30	58

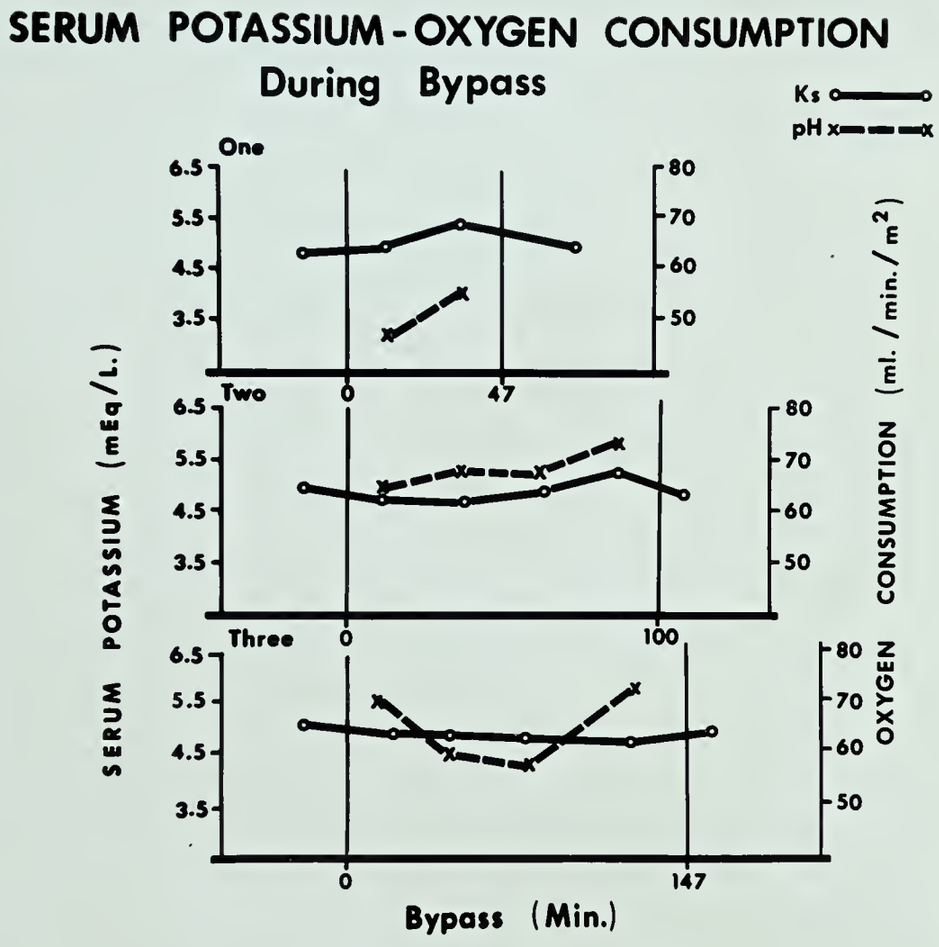

SERUM POTASSIUM - OXYGEN CONSUMPTION
During Bypass
Ks
pH
One
Two
Three
6.5
5.5
4.5
3.5
80
70
60
50
0
47
100
147
SERUM POTASSIUM (mEq/L.)
OXYGEN CONSUMPTION (ml./min./m²)
Bypass (Min.)

Figure 5

drop of 38 ml./min./m^2 in case ten had been excluded.

The oxygen consumption appeared to seek a predetermined level during bypass. From interval one to interval three the range of values narrowed progressively. In interval four, when changes in the blood flow and saturation levels were most marked, the range increased. In Cases 17 and 23 the oxygen consumption dropped 18 and 26 ml./min./m^2 between two consecutive intervals. The serum potassium changes in all these cases were mixed, showing no consistent change in magnitude or direction (Table XVIII). The low 'r' value (0.13) means that when results from the same samples were used, the oxygen consumption was not predictably related to the serum potassium level. This finding is consistent with the results of Clarke (14) who found that the arterial and venous oxygen saturations take fifteen minutes to equilibrate with changes in the blood flow. It does not rule out the possibility that the serum potassium may have a better correlation with the oxygen consumption several minutes after the change.

The Effect of Hypothermic Bypass

The five hypothermic bypass cases, which were all in Groups II and III, had mean interval oxygen consumptions of 72, 54, 57 and 69 ml./min./m^2.

The bypass potassium patterns of the hypothermic patients were similar to the nonhypothermic patterns (Table XIX)

TABLE XVIII

OXYGEN CONSUMPTION DURING BYPASS

(ml./min./m^2.)

Case No.	Group	Surface Area (m^2.)	Bypass Oxygen Consumption Levels			
			1	2	3	4
8	II	1.82	65	66	64	56
9	II	1.78	45	51	53	48
10	III	1.47	103	65	52	69
11	II	1.85	60	71	72	46
12	II	2.06	44	55	60	57
13	III	2.14	55	53	71	82
14	I	1.69	63	--	--	--
15	II	1.76	64	58	51	55
16	I	1.60	50	48	--	--
17	I	1.63	62	44	--	--
18	I	1.47	28	41	--	--
19	II	1.75	39	49	71	99
20	II	2.08	72	73	62	68
21	I	1.81	54	82	--	--
22	II	1.94	74	69	71	79
23	II	1.66	86	89	63	106
24	II	1.67	77	74	81	98
25	II	1.90	61	66	70	77
26	III	2.16	59	70	62	69

TABLE XIX

SERUM POTASSIUM LEVELS DURING HYPOTHERMIC BYPASS

(in mEq/l)

Group	Case No.	Bypass					
		Pre	1	2	3	4	Post
III	7	6.0	5.8	5.5	4.7	4.3	3.9
III	10	5.0	4.6	4.6	4.6	5.0	4.9
II	12	5.0	4.9	4.3	5.2	5.4	4.2
II	15	4.4	4.7	4.1	4.3	4.8	4.3
II	25	4.5	3.9	4.5	4.5	4.3	3.8
Means		5.0	4.8	4.6	4.7	4.8	4.2
Non-hypothermic Means		4.8	4.7	--	--	--	4.6

except for case seven where the washout of potassium, which accumulated during the prebypass hypotensive period, decreased the bypass level by 1.5 mEq/l.

All five patients developed ECG abnormalities during bypass which invalidated any conclusions based on the incidence of operative arrhythmias. Two atrial fibrillations and three ventricular fibrillations arose. The two atrial arrhythmias spontaneously reverted to normal rhythms upon rewarming. The other three patients required at least one electrical defibrillation.

Other Factors

The Blood Balance During Surgery

In twenty patients, pre and postoperative blood volume determinations were made using isotope dilution techniques. (Tables XX, XXI, XXII). The average patient lost 181 ml. red cells and gained 33 ml. of plasma for an average net blood volume loss of 147 ml. These results have been compared with the blood balance figures of the anesthetist. (Table XXIII). The anesthetist calculated the balance between the blood lost into the suction system, swabs, drapes and pump system after bypass, and the amount of donor blood administered intravenously. The isotope method on the average gave a 150 ml. larger postoperative blood volume than did the anesthetists' method. Both methods indicated that the postoperative blood volume was

TABLE XX

PREOPERATIVE BLOOD VOLUMES

Case No.	Red Cell Volume		Plasma Volume		Total Blood Volume	
	cc.	cc/Kg.	cc.	cc/Kg.	cc.	cc/Kg.
1	1770	26	2820	41	4590	67
2	1980	24	2603	31	4583	55
3	--	--	--	--	--	--
4	--	--	--	--	--	--
5	1144	24	2088	44	3232	68
6	--	--	--	--	--	--
7	1920	27	3034	43	4954	70
8	1583	23	2243	33	3826	56
9	1835	29	2790	44	4625	73
10	--	--	--	--	--	--
11	1930	27	2505	35	4435	62
12	1890	23	3165	39	5055	62
13	2220	24	3640	41	5860	65
14	1440	24	2672	45	4112	69
15	2550	34	3120	42	5670	76
16	1586	29	2025	37	3611	66
17	1378	26	1965	36	3343	62
18	--	--	--	--	--	--
19	1546	22	2600	37	4146	59
20	1723	20	2745	32	4468	52
21	1633	24	2355	34	3988	58
22	1955	24	3100	38	5055	62
23	1905	32	2010	33	3915	65
24	1278	19	2540	38	3818	57
25	1570	23	3123	45	4693	68
26	2590	30	4540	53	7130	83
Means	1782	26	2747	40	4529	66

TABLE XXI

POSTOPERATIVE BLOOD VOLUMES

Case No.	Red Cell Volume		Plasma Volume		Total Blood Volume	
	cc.	cc/Kg.	cc.	cc/Kg.	cc.	cc/Kg.
1	1810	26	3200	46	5010	72
2	1670	20	2840	34	4510	54
3	--	--	--	--	--	--
4	--	--	--	--	--	--
5	1138	24	2067	43	3205	67
6	--	--	--	--	--	--
7	1890	27	3670	52	5560	79
8	1339	20	1685	24	3024	44
9	1305	21	2545	40	3845	61
1o	--	--	--	--	--	--
11	1925	27	2780	39	4705	66
12	2200	27	3800	47	6000	64
13	1640	18	3355	37	4995	56
14	1350	23	1850	31	3200	54
15	1700	23	2990	40	4690	63
16	1055	19	2227	40	3282	59
17	1220	23	2235	41	3455	64
18	--	--	--	--	--	--
19	1400	20	2342	33	3742	53
20	1480	18	2760	32	4240	50
21	1226	18	2790	40	4016	58
22	1950	24	3260	40	5210	64
23	1605	27	2210	36	3815	63
24	1410	21	2735	41	4145	62
25	1810	26	3130	45	4940	71
26	2500	30	3900	45	6460	75
Means	1601	23	2780	41	4381	64

TABLE XXII

BLOOD VOLUME CHANGES DURING SURGERY

Case No.	Red Cell Volume		Plasma Volume		Total Blood Volume	
	cc.	cc/Kg.	cc.	cc/Kg.	cc.	cc/Kg.
1	+ 40	+ .6	+ 380	+ 5.4	+ 420	+ 6.0
2	- 310	- 3.7	+ 237	+ 2.9	- 73	- .8
3	-	-	-	-	-	-
4	-	-	-	-	-	-
5	- 6	- .1	- 20	- .4	- 26	- .5
6	-	-	-	-	-	-
7	- 30	- .4	+ 636	+ 9.0	+ 606	+ 8.6
8	- 244	- 3.5	- 558	- 8.2	- 802	-11.8
9	- 530	- 8.4	- 245	- 3.8	- 775	-12.3
10	-	-	-	-	-	-
11	- 5	- .1	+ 275	+ 3.8	+ 270	- 3.8
12	+ 310	+ 3.8	+ 635	+ 7.8	+ 945	+11.7
13	- 580	- 6.5	- 285	- 3.2	- 865	- 9.7
14	- 90	- 1.5	- 822	-13.9	- 912	-15.4
15	- 850	- 11.4	- 130	- 1.8	- 980	-13.2
16	- 531	- 9.7	+ 202	+ 3.7	- 329	- 6.0
17	- 158	- 2.9	+ 270	+ 5.0	+ 112	+ 2.1
18	-	-	-	-	-	-
19	- 147	- 2.1	- 258	- 3.7	- 405	- 5.8
20	- 243	- 2.8	+ 15	+ .2	- 228	- 2.7
21	- 407	- 5.9	+ 445	+ 6.4	+ 32	+ .5
22	- 5	- .1	+ 160	+ 2.0	+ 155	+ 2.6
23	- 300	- 5.0	+ 200	+ 3.3	- 100	- 1.7
24	+ 132	+ 2.0	+ 195	+ 2.9	+ 327	+ 4.9
25	+ 220	+ 3.9	+ 7	+ .1	+ 277	+ 4.0
26	- 90	- .6	- 640	- 7.7	- 730	- 8.4
Means	- 181	- 2.6	+ 33	+ 0.5	- 147	- 2.1

TABLE XXIII

NET BLOOD BALANCE

	METHOD I Isotope Calculation			METHOD II Anesthetist Calculation			METHOD I minus METHOD II
	Red Cell Volume Change	Plasma Volume Change	Net Volume Change	Blood Loss	Blood Gain	Net Change	
1	+40	+380	+420	2913	3150	+237	-17
2	-310	+237	-73	2896	2500	-396	+326
3	-	-	-	3904	3500	-404	-
4	-	-	-	1820	2200	+380	-
5	-6	-20	-26	3050	3000	-50	+24
6	-	-	-	3290	3575	+280	-
7	-30	+636	+606	3700	3800	+100	+510
8	-244	-558	-802	1750	1000	-750	-50
9	-530	-245	-775	2515	1750	-765	-10
10	-	-	-	1765	1005	-760	-
11	-5	+275	+270	2264	2000	-264	+534
12	+310	+635	+945	3478	4000	+522	+423
13	-580	-285	-865	14950	14350	-600	-265
14	-90	-822	-912	1961	1500	-461	-451
15	-850	-130	-980	5700	5000	-700	-180
16	-531	+202	-329	1254	500	-754	+425
17	-158	+270	+112	890	500	-292	+404
18	-	-	-	1195	300	-895	-
19	-147	-258	-405	3100	2500	-600	+195
20	-243	+15	-228	2170	2000	-170	-58
21	-407	+445	+32	932	250	-682	+714
22	-5	+160	+155	2300	2100	-200	+355
23	-300	+200	-100	2738	2000	-738	+638
24	+132	+195	+327	2384	2250	-134	+461
25	+220	+7	+277	3780	4000	+220	+57
26	-90	-640	-730	7605	7000	-605	-125

usually smaller than the preoperative blood volume.

The differences between the isotope and the anesthetists' methods resulted from the presence of Ringer's lactate and ACD solution in the plasma one hour after surgery, which the anesthetists assumed had passed extravascularly. Minor differences in individual cases may have been due to hematocrit differences between the fluid lost and the fluid gained. In the short procedures, blood was lost primarily before and after bypass at higher hematocrits than in the transfused blood. This resulted a net red cell loss. In the longer procedures, more blood was lost during or at the end of bypass at a lower hematocrit than in the donor blood. This resulted in a net red cell gain.

Individual fluid therapy was usually necessary. In most cases, a deficit of approximately 500 ml. was allowed to arise during bypass and a blood balance was achieved only by the end of surgery. This avoided an overload problem which could have resulted in a small patient if the pump fluid were transfused into a fully balanced cardiovascular system. Some anesthetists terminated surgery with an estimated 100-300 ml. deficit to take into account the Ringer's lactate still present in the intravascular space.

Case 13 required thirty transfusions to replace blood lost from the base of the aortic arch. Because of the multiple exchange transfusions, the recipient's hematocrit

and the donor's hematocrit were similar and, despite these large volume changes, the two methods were within two hundred cc. in their estimation of the final blood volume.

Case 21, a Group I patient, had a net red cell loss (anesthetist's method) of 300 ml. excluding losses into traumatized areas. This was close to the 407 ml. red cell loss calculated by the isotope method. The net non-red cell fluid additions totalled 1545 ml. of which approximately 125 ml. contained albumin. The RISA plasma volume increased 439 ml. suggesting that one hour after surgery approximately 1100 ml. of fluid had passed extracellularly and into the urine. One thousand mls. of urine were formed within the next 24 hours. Interestingly, the hematocrit did not reflect the twenty-five percent decrease in the red cell volume and twenty percent increase in the plasma volume. It dropped from a prebypass 42 to a postbypass 39 instead of the predicted 31. His F cells (18) or ratio of whole body hematocrit to large vessel hematocrit was 0.76 which was the lowest in the series. This suggests that considerable amounts of plasma were pooled peripherally.

The Net Fluid Balance During Surgery

The net fluid balance and the net lactate and saline balance for each patient are given in Table XXIV. The net fluid balance was positive in every case and averaged

TABLE XXIV

NET OPERATIVE FLUID AND LACTATE AND SALINE GAIN

Case No.	Net Operative Fluid Addition (Excluding Insensible Losses)		Net Operative Lactate and Saline Addition	
	cc.	cc/Kg.	cc.	cc/Kg.
1	2565	37	2600	38
2	1924	23	2450	29
3	1556	27	2100	36
4	1960	31	1700	28
5	1445	30	1650	34
6	1997	40	2000	40
7	2600	37	2800	39
8	1485	22	2500	37
9	1085	17	2300	36
10	1037	22	1900	40
11	1656	16	2140	30
12	3522	43	3300	41
13	2570	29	3400	39
14	844	14	1500	25
15	2265	31	3300	44
16	526	10	1400	25
17	1048	19	1600	30
18	390	8	1200	24
19	2200	31	3100	44
20	1780	21	2070	24
21	782	12	1580	28
22	1635	20	2250	27
23	1582	26	2500	41
24	1446	22	1540	23
25	1990	28	2100	30
26	1965	23	2700	31
Means	1680	25	2220	33

1699 ml. or 25 cc/Kg, while the lactate and saline additions averaged 26, 34 and 37 cc/Kg.

Case 12, a Group III patient, had the largest net fluid overload, (3520 cc or 43 cc/Kg) of which 3300 cc were lactate and saline. His blood volume increased by 945 ml. Only 300 ml. of urine were formed during surgery because blood replacements could not match the losses. The excess fluid was excreted in the 2390 ml. of urine formed during the first postoperative day.

Case 18 had the lowest net fluid increase, 250 ml. or 5 cc/Kg. Blood replacements to this Group I patient were 900 ml. behind losses at the end of bypass, so that transfusion of the 1000 ml. of lactate prime did not result in a fluid overload as monitored by the CVP or ECG.

The Urine Formation During Surgery

Operative urine volumes ranged from 60 to 450 ml. (Table XXV) or from 1.0 to 7.1 cc/Kg. In Groups I, II and III the average urine formation was 3.2, 3.5 and 3.2 cc/Kg. despite the wide variations in net lactate additions, perfusion durations, total operative durations, net fluid balance and grams of mannitol received per kilogram of body weight. Breakdown of the operative urine formation into prebypass, bypass and postbypass intervals was attempted (Table III) but discontinued after case seventeen because of collection problems. A visual pattern however was noticed. The urine formed before bypass was either small and con-

TABLE XXV

URINE FORMATION

Case No.	Operative		Postoperative (24 hours)	
	cc.	cc/Kg.	cc.	cc/Kg.
1	272	3.9	520	7.5
2	130	1.6	820	10.0
3	140	2.4	1100	19.0
4	120	2.0	550	9.1
5	155	3.3	890	18.0
6	213	4.0	died	--
7	300	4.2	died	--
8	265	4.0	1936	28.0
9	450	7.1	570	9.1
10	203	4.0	590	12.0
11	220	3.1	750	10.4
12	300	3.1	2390	40.0
13	230	2.6	1920	21.8
14	195	3.2	800	13.5
15	335	4.5	925	12.5
16	120	2.2	580	10.5
17	260	5.0	540	10.0
18	60	1.2	730	14.6
19	300	4.3	570	8.1
20	120	1.4	700	8.1
21	210	3.0	950	13.8
22	415	5.1	560	6.8
23	180	3.0	700	11.7
24	70	1.0	825	12.3
25	330	4.7	550	8.0
26	235	2.7	800	9.3
Means	217	3.4	885	14.3

centrated or nonexistent. With the onset of bypass and the addition of 25 grams of mannitol, an abundant flow of clear urine was produced although it usually stopped by the end of bypass. After the pump fluid was transfused, a variable amount of urine was formed primarily during the first part of the postbypass period.

The urinary potassium content and the urine volume changed together ($r = 0.77$, Table V). Because sodium retention reduced the urine sodium content and because hyperventilation produced an alkalotic extracellular fluid, the potassium ion constituted the primary cation in the urine. Hence its close correlation with the urine volume. Comparison of the urine formation and urine potassium content with the volume of Ringer's lactate given during surgery gave 'r' values of 0.51 and 0.54. The same correlations with the net fluid volume addition gave 'r' values of 0.18 and 0.32. This indicates that the total lactate load was one factor influencing the operative urine formation and potassium excretion but the net fluid volume addition was not a factor. A closer examination of the total lactate load and the rate of urine formation and potassium excretion during and immediately after bypass would be necessary to further clarify this relationship.

Operative and Postoperative Arrhythmias

Twelve patients developed arrhythmias during or after surgery (Table IV). One was on diuretics and digitalis,

four were on digitalis and one was on diuretics preoperatively. Eight of the twelve arrhythmias occurred during surgery. Five occurred in patients undergoing mild hypothermic bypass, two of which spontaneously reverted to normal ECG patterns upon rewarming.

All arrhythmias developed when serum potassium levels were normal. Conversely, no arrhythmias developed when serum potassium concentrations were abnormal. It would appear that arrhythmias and abnormal serum potassium levels did not occur together and therefore were not the result of fluctuations in the serum potassium.

Specific Problems

Methods of Analysis

Body adjustments must be instantaneous for any correlation to exist between two changing parameters. If the adjustment is delayed no correlation can be shown unless sampling intervals are sufficiently frequent to illustrate the non-instantaneous relationship. A low correlation coefficient does not rule out the existance of a potential cause and effect relationship, nor does a high correlation prove that such a relationship exists. It only proves that the two parameters change in the same direction in the same proportions.

Where single examples have been discussed in detail, implications have been made. To conclude that they repre-

sent all cases is fallacious, certainly by statistical analysis.

Analysis of Patients

Four human variables exist in the operating theatre: the anesthetist, the pump technician, the surgeon and the patient. The first three control the quality of anesthesia, the fluid balance, the perfusion rate and the functional recovery of the heart. With the addition of patient differences in age, sex, cause and duration of disease, type of treatment and pre and postoperative potassium status, wide variations in the results are guaranteed. To pinpoint areas for further qualitative and quantitative research, longitudinal studies must be made which use each patient as his own control.

Hydrogen ion Fluctuations

The usual practice was for the anesthetist to hyperventilate the patient during surgery. If this was done consistently, then the serum pH during cardiopulmonary surgery should have been constant from patient to patient. Complications arose when each anesthetist used a different "normal" which resulted in a different pH and hence a different serum potassium environment.

Fluid Balance

Because rapid and accurate assessment of the net fluid or blood balance at any particular moment is difficult, the blood balance after transfusion of the pump fluid was often assumed to be zero. In older patients, this policy became particularly important because a variable amount of lactate was always present in the circulation after bypass. When this fluid passed extravascularly the patient could be left with an inadequate blood volume several hours after surgery. Even if volume titration could be achieved, the hematocrit differences between donated and lost blood made red cell and plasma titration impossible.

To measure the actual red cell and plasma volume fluctuations after surgery, a double isotope dilution technic with single sampling after equilibration was used. This method did not take into account any losses of tagged red cells or iodinated albumin during the equilibrium period. Losses into traumatized areas or drainage tubes can only be measured by serial sampling with semilogarithmic extrapolation back to the original injection time. Since this was not done, the blood volume in some cases, may be slightly overestimated.

Where large transfusions were necessary, the serum calcium, pH and the body temperature were depressed and large unmeasured quantities of free potassium and ACD solution may have been added. These variables were not measured and their influences, if any, on the serum potassium

is unknown.

Potassium Studies

The twelve hour half life of Potassium-42 (58) and the twenty-four hour equilibration period required to measure the total body potassium (77,86) prevent determination of the preoperative total body potassium. To determine the net operative change in this undefined pool, all potassium additions and losses must be measured. Because the free potassium concentrations in the added and lost solution were within the narrow 3-6 mEq/l range, an average net fluid gain of 1700 cc (Table XXIV) resulted in a maximum extracellular fluid volume addition of 10 mEq.of potassium or a serum potassium change of 0.3 mEq/l in a 75 kilogram patient. While some of this potassium may have been excreted in the urine, it did not account for all the urinary potassium. Therefore, some of the potassium excreted must have had an intracellular source.

Arrhythmias

To establish a cause and effect relationship between the serum potassium and any arrhythmias, measurement of the intracellular and extracellular potassium concentrations at the moment the arrhythmia developed would be necessary. In lieu of such measurements it has been assumed that the serum potassium level is synonymous with the extra-

cellular potassium concentration and that the intracellular potassium concentration is constant (115). Furthermore, because similar but abnormal potassium concentrations have different effects on the ECG pattern, conclusions based on the arrhythmias-serum potassium relationship in this study are questionable and difficult to interpret.

General Outcome

Four patients subsequently underwent reoperations. This included Case 15 who hadan ASD that was operated upon previously. Cases 13 and 7 underwent immediate reoperations for bleeding at the base of the grafted aortic arch and for bleeding from the myocardial puncture sites respectively. Case 13 survived. Case 7 died from uncontrolled myocardial bleeding and anuria on postoperative day 2. Case 25 underwent a second operation for recurrent aortic regurgitation, one month after the first procedure, but died one day later. Case 6, the third patient to pass away, died twenty-four hours after surgery from atrial fibrillation, congestive heart failure, pyrexia of unknown origin and Isuprel resistant hypotension.

Four arrhythmias occurred postoperatively. One patient, with atrial fibrillation preoperatively, continued fibrillating. Another patient developed an atrial fibrillation. A third developed numerous extrasystoles uncontrolled by Pronestyl. A fourth patient arrested on the second post-

operative day having had a continuous bigeminal pulse since surgery. Resuscitation was successful and the patient was discharged, markedly improved.

Nine of the twelve preoperative digitalized patients had digitalis reordered within ten days of surgery, along with seven other patients.

Unfortunately, many patients were discharged to the care of physicians in distant centres, making survival figures incomplete.

CHAPTER IV

SUMMARY AND CONCLUSIONS

The serum potassium pattern in twenty-six consecutive adult cardiopulmonary operations at the University of Alberta Hospital has been reviewed and analysed. Twelve of the twenty-six patients were on digitalis therapy preoperatively. Seven patients were on diuretics including five on both digitalis and diuretics. The serum potassium differences which existed between the patients on preoperative medication and the patients not on medication vanished as surgery progressed.

During the prebypass period, the mean serum potassium level rose 0.9 mEq/l from a preoperative base of 3.9 mEq/l. After a slight 0.1 mEq/l drop with the onset of bypass, the mean rose 0.3 mEq/l to a late bypass peak of 5.0 mEq/l, then dropped to 4.5 after bypass, rose to 4.7 on day one, and dropped to 3.8 mEq/l on day five.

The prebypass rise in the serum potassium resulted from an imbalance between the urinary potassium excretion and the release of cell potassium from traumatized cells. In the four patients, whose prebypass blood pressures dropped below 75 mmHg under halothane anaesthesia, the serum potassium rose 1.6 mEq/l. In the patients who did not have a hypotensive period before bypass, the serum potassium rose 0.9 mEq/l. The 0.1 mEq/l drop in the serum

potassium onto bypass is probably due to plasma potassium dilution by the priming solution.

The mean potassium concentrations rose in three groups which were formed when the twenty-six patients were grouped according to their bypass times. The coefficient of correlation between the change in the potassium correlation and the duration of perfusion was 0.18. By excluding seven hypotensive patients and by using the first bypass sample of each patient as a baseline, the 'r' value rose to 0.48. It is therefore concluded that the duration of perfusion was one factor that affected the serum potassium level during bypass. By plotting the bypass arterial pCO_2, HCO_3 and pH values on an acid-base nomogram a respiratory alkalosis was noted to exist in most bypass samples. It most commonly originated from the low CO_2 concentration in the oxygen-anesthetic gas mixture. In comparing the oxygen consumption and the bypass serum potassium levels, a coefficient of 0.08 resulted. This indicated that oxygen consumption affects the serum potassium less than the pH.

Gross estimation of the net fluid balance during surgery indicated that all patients received a positive fluid balance ranging from five to forty-one cc/kg. A considerable amount of this fluid was in the form of Ringer's lactate. The Ringer's lactate additions during surgery bore some correlation with the urine volume ($r = 0.51$), and the urinary potassium ($r = 0.52$). Urinary potassium correlated even better with the urine volume ($r = 0.78$). The

average red cell and plasma volume changes in the twenty patients so studied were 181 ml. and 433 ml.

Eight arrhythmic patterns developed during surgery, including five in the five patients who underwent bypass at mild hypothermic levels. The abnormal rhythms included two atrial fibrillation patterns which spontaneously reverted upon rewarming and three ventricular fibrillations which required at least one defibrillation attempt. Four patients developed atrial conduction abnormalities postoperatively and one patient arrested, but was successfully resuscitated.

Of the twelve patients on digitalis preoperatively, nine were redigitalized postoperatively as were six others.

Three patients are known to have died in the three month follow-up period. One died after a reoperation on the same day as the initial procedure. A second case died following a reoperation for aortic incompetance around the artificial valve. The third patient died of congestive heart failure and pulmonary embolization two days after surgery.

In conclusion, any prebypass hypotension with systemic pressures below 75 mmHg., reduced renal potassium excretion and retained potassium released from traumatized cells. The net movement of potassium into the remaining cells was insufficient to limit an elevation in the extracellular potassium concentration. During bypass, the most significant serum potassium determinant was the duration of bypass. The pH or its respiratory and metabolic components,

and the oxygen consumption did not influence the serum potassium when judged by their low correlation coefficients. The total Ringer's lactate load has some effect on the total urine volume. The urine volume and the urine potassium content bore an even closer relationship with the serum potassium. The arrhythmias which developed were not associated with abnormal serum potassium levels. Nor were the abnormal serum potassium levels associated with any arrhythmias.

While the prebypass systemic blood pressure, the duration of perfusion, the volume of urine and the quantity of potassium excreted during the operation all influenced the serum potassium. The capacity of the body to limit fluctuation in this level beyond the normal range was most remarkable.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Abramson, E., Arky, R., Diabetic Acidosis with Initial Hypokalemia. J.A.M.A. 196:401, 1966.

2. Andersen, M.N., Norberg, B., Senning,A., Studies of Liver Function during Extracorporeal Circulation with low-flow Rate. Surgery 43:397, 1958.

3. Astrup, P., Siggaard, Andersen, O., Jorgensen, K., Engel, K., The Acid-Base Metabolism - A New Approach. Lancet I:1035, 1960.

4. August, J.T., Nelson, D.H., Thorn, G.W., Aldosterone. New Eng. J. Med.,259:917,967, 1958.

5. Barnard, M.S., Saunders,S.J., Eales, L., Barnard, C.N., Hypokalemia during Extracorporeal Circulation. Unpublished data.

6. Beall, A.C., Cooley, D.A., Physiological Studies During Cardiopulmonary Bypass Eliminating Heparinized Blood. Dis. Chest 47:7, 1965.

7. Bell, R.E., Personal Communication.

8. Berliner, R.W., Levinsky, N.G., Davidson, O,G., Eden, M., Dilution and Concentration of the Urine and the Action of Antidiuretic Hormone. Am. J. Med. 24:730, 1958.

9. Bernstein, J., Untersuchungen zur Thermodynamik der biologischen Strome, Pflug. Arch des Physiol. 92: 521, 1902. Quoted in Cell K, Kernan, R.P., Butterworths, Washington, 1965, p. 59.

10. Brazier, M.A.B., Some effects of Anaesthesia on the Brain. Br. J. Anaesth. 33:194, 1961.

11. Buchanan, D.I., Personal Communication.

12. Campbell, D.J. Personal Communication.

13. Cheng, H.C., Kusonoki, C.T., Busher, L.H., McElvein, R.B., Blake, D.A., Study of Oxygen Consumption during Extracorporeal Circulation. Trans. Am. Soc. Artificial Internal Organs 5:273, 1959.

14. Clark, L.C., Optimal Flow Rate in Perfusion. Extracorporeal Circulation, ed. Allen, J.G., Thomas, Springfield, Ill. 1958, p. 150.

15. Clowes, G.H.A., Extracorporeal Maintenance of Circulation and Respiration. Physiol. Rev. 40:826, 1960.

16. Conway, E.J., Nature and Significance of Concentration Relations of Potassium and Sodium Ions in Skeletal Muscle. Physiol. Rev. 37:84, 1957.

17. Craig, A.B., Observations on Epinephrine and Glucagon-induced Glycogenolysis and Potassium Loss in the Isolated Perfused Frog Liver. Amer. J. Physiol. 193:425, 1958.

18. Dagher, F.S, Lyons, J.H., Finlayson, D.C., Shamsai, J., Moore, F.D., Blood Volume Measurement: a critical study. in Advances in Surgery, Yearbook Medical Publishers, Chicago, 1965, p. 69.

19. Deutsch, S., Linde, H.W., Dripps, R.D., Price, H.L., Circulatory and Respiratory Actions of Halothane in Normal Man. Anesthesiology 23:631, 1962.

20. DeWall, R.A., Lillehei,C.W., Simplified Total Body Perfusion. J.A.M.A. 179:430, 1962.

21. DeWall, R.A., Lillehei, R.C., Perfusion for Open-Heart Surgery Requiring only 5% Dextrose in Water for Pump Priming. Surg. Clin. N. Amer. 44:253, Feb.1964.

22. DeWall, R.A., Lillehei, R.C., Sellers, R.D., Hemodilution Perfusions for Open Heart Surgery, (Use of 5% Dextrose in Water for the Priming Volume). New Eng. Jour. Med. 266:1078, 1962.

23. Drury, D.R., Wick, A.N., Epinephrine and Carbohydrate Metabolism. Amer. J. Physiol. 194:465, 1958.

24. D'Silva, J.L., Action of Adrenaline on the Serum Potassium. J. Physiol.90:303, 1937.

25. Dunham, E.T., Glynn, I.N., Adenosinetriphosphatase Activity and the Active Movements of Alkali Metal Ions. J. Physiol. 156:274, 1961.

26. Earley, L.E., Orloff, J., Thiazide Diuretics. Ann. Rev. Med. 15:149, 1964.

27. Ebert, P.A., Jude, J.R., Gaertiner, R.A., Persistent Hypokalemia Following Open-Heart Surgery. Circulation 31 Suppl. I:137, 1965.

28. Ebert, P.A., Morron, A.G., Austen,W.G., Clinical Studies on the Effect of Extracorporeal Circulation on Myocardial Digoxin Concentration. Amer. J. Cardiol. 11:201, 1963.

29. Edelman, I.S., Bogoroch, R., Porter, G.A., On the Mechanism of Action of Aldosterone on Sodium Transport: the role of protein synthesis. Proc. Nat. Acad. Sci. U.S.A., 50:1169, 1963.

30. Eichenholz, A., Mulhausen, R.O., Blumentals, A., Acid-Base Balance Determinations in Clinical Practice. Postgrad. Med. 38:A68, 1965.

31. Ellis, S., The Metabolic Effect of Epinephrine and Related Amines. Pharmacol. Rev. 8:485, 1956.

32. Finder, A.G., Boyme, T., Shoemaker, W.C., Relationship of Hepatic Potassium Efflux to Phosphorylase Activation Induced by Glucagon. Amer. J. Physiol. 206:738, 1964.

33. Finkenstaedt, J.T., Ruiz-Guinazu, A., Moreau, L., Morrison, R.S., Merrill, J.P., Experimental Acute Potassium Depletion in Dogs. Clin. Sci. 16:171, 1957.

34. Fordham, R.M.M., Hypoxemia after Aortic Valve Surgery under Cardiopulmonary Bypass. Thorax 20:505, 1965.

35. Frank, G.B., Calcium and the Initiation of Contraction. Circ. Res. 15 Suppl. II:54, 1964.

36. Glynn, I.M., The Action of Cardiac Glycosides on Ion Movements. Pharmacol. Rev. 16:381, 1964.

37. Goodman, L.S., Gillman, A., The Pharmacological Basis of Therapeutics, Third Edition. Macmillan, New York, 1966, p. 400.

38. Ibid, p. 492.

39. Ibid, p. 1619.

40. Goth, A., Medical Pharmacology, Third Edition. C.V. Mosby, St. Louis, 1966, p. 324.

41. Greenman, L., Mateer, F.M., Gow, R.C., Peters, J.H., Danowski, T.S., Some Observations on the Development of Hypokalemia during Therapy of Diabetic Acidosis in juvenile and young adult subjects. J. Clin. Inv. 28:409, 1952.

42. Guyton,A.C., Textbook of Medical Physiology, Third Edition. Saunders, Philadelphia, 1966, p. 983.

43. Hajdu, S., Leonard, E., Cellular Basis of Cardiac Glycoside Action. Pharmacol. Rev. 11:173, 1959.

44. Hagen, J.H., Hagen, P.B., Action of Adrenalin and Noradrenalin on Metabolic Systems. in Action of Hormones on Molecular Processes, ed. Litwak, G., Kretchovsky, D., John Wright and Sons, Inc., 1964.

45. Hammarsten, J.F., Ginn, H.E., Smith, W.V., in Clinical Metabolism of Body Water and Electrolytes, Saunders, Philadelphia, 1963, p. 569.

46. Hodgkin, A.L., Ionic Movements and Electrical Activity In Giant Nerve Fibres. Proc. roy. Soc. B148:1, 1958.

47. Hodgkin, A.L., Huxley, A.F., A Quantitative Description of the Membrane Current and its Application to Conduction and Excitation in Nerve. J. Physiol.117:500, 1952.

48. Hodgkin, A.L., Katz, B., The Effect of Sodium Ions on the Electrical Activity of the Giant Axon of the Squid. J. Physiol. 108:37, 1949.

49. Hoel, P.G., Elementary Statistics. Second Edition, John Wiley, & Sons, Inc., New York, 1966, p. 27.

50. Ibid, p. 173.

51. Ibid, p. 198.

52. Hoffman, J.F., The Active Transport of Sodium by Ghosts of Human Blood Cells, J. gen. Physiol. 45:837, 1962.

53. Holland, W.C., Ion Distribution and Myocardial Metabolism as Affected by Cardiac Glycosides. Circ. Res. 15 Suppl. II:85, 1964.

54. Holliday, M.A., Egan, T.J., Dehydration, Salt Depletion, and Potassium loss. Ped. Clin. N. Amer. 11:871, 1964.

55. Jamison, R.A., Kay, A.W., A Textbook of Surgical Physiology. Livingstone, Edinburgh and London, 1959, p. 49.

56. Johansson, B.W., Sievers, J., The Effects on Serum Concentrations of Sodium Potassium and Chloride during long term treatment with some Diuretics. Angiology 17:134, 1966.

57. Joyce, C.R.B., Weatherall, M., Cardiac Glycosides and the Potassium Exchange of Human Erythrocytes. J. Physiol. 127:33P, 1955.

58. Kaman, M.D., Isotope Tracers in Biology. Academic Press Inc., New York, 1957, p. 363.

59. Keele, C.A., Neil, E., Samson Wright's Applied Physiology. Eleventh Edition. Oxford University Press, London, 1965, p. 125.

60. Kernan, R.P., Cell K. Butterworths, Washington, 1965,p.19.

61. Kernan, R.P., The Role of Lactate in the Active Excretion of Sodium by Frog Muscles, J. Physiol. 162:129, 1962.

62. Kinney, J.M., Moore, F.D., in Clinical Metabolism of Body Water and Electrolytes. ed. Bland, J.G., Saunders, Philadelphia, 1963, p. 337.

63. Laragh, J.G., Capeci, N.E., The Effect of Administration of Potassium Chloride on Serum Sodium and Potassium Concentration. Amer. J. Physiol. 180:539, 1955.

64. Leaf, A., Santos, R.F., Physiologic Mechanisms in Potassium Deficiency. New Eng. J. Med. 264:335, 1961.

65. Lee, K.S., Effect of Ouabain on Glycerol-extracted fibres from Heart Containing a "relaxing factors". J. Pharmacol. Exp. Ther. 132:149, 1961.

66. Leibman, J., Edelman, I.S., Interrelations of Plasma Potassium Concentrations, Plasma Sodium Concentrations Arterial pH, and Total Exchangeable Potassium. J. Clin. Inv. 38:2176, 1959.

67. Liddle, G.W., Duncan, L.E. Jr., Bartter, F.C., Dual Mechanism Regulating Adrenocortical Function. in Man. Amer. J. Med. 21:380, 1956.

68. Litchfield, J.A., Gaddie, R., The Measurement of the Phase Distribution of Water and Electrolytes in Skeletal Muscle by the Analysis of Small Samples. Clin. Sci. 17:483, 1958.

69. Litwin, M.S., Panico, F.G., Rubin, C., Harken, D.E., Moore, F.D., Acidosis and Lactacidemia in Extracorporeal Circulation: Significance of perfusion flow rate and the relation of perfusion respiratory alkalosis. Ann. Surg. 149:188, 1959.

70. Lockey, E., Ross, D.N., Longmore, D.B., Sturridge, M.F., Potassium and Open-Heart Surgery. Lancet I:671, 1966.

71. Long, C.N.H., Smith, O.K., Fry, E.G., Actions of Cortisol and Related Compounds on Carbohydrate and Protein Metabolism. in CIBA Foundation Study Group No. 6: Metabolic Effects of Adrenal Hormones, ed. Wolstenholme, G.E.W., O'Conner, M., Churchill, London, 1960, p. 4.

72. Luttgau, H.C., Niedergerke, R., The Antagonism between Calcium and Sodium ions on the Frog's Heart. J. Physiol. 143:486, 1958.

73. McCann, S.M., The ACTH-releasing Activity of Extracts of the Posterior Lobe of the Pituitary in Vivo. Endocrinology 60:664, 1957.

74. McGoon, D.C., Moffitt, E.A., Theye, R.A., Kirklin, J.W., Physiologic Studies during High Flow, Normothermia, Whole Body Perfusion. J. Thor. Cardiov. Surg. 39:275, 1960.

75. Maizels, M., Remington, M., Cation Exchanges in Human Erythrocytes. J. Physiol. 145:641, 1959.

76. Miller, B.J., Gibbon, J.H. Jr., Gibbon, M.H., Recent Advances in Development of Mechanical Heart and Lung Apparatus. Ann. Surg. 134:694, 1951.

77. Miller, C.E., Remenchik, A.P., Problems involved in Accurately Measuring the Potassium Content of the Human Body. Ann. N.Y. Acad. Sci. 110:175, 1963.

78. Moore, F.D., Metabolic Care of the Surgical Patient. Saunders, Philadelphia, 1959, p. 7 - 9.

79. Ibid, p. 32 - 38.

80. Ibid, p. 62.

81. Ibid, p. 109 -117.

82. Moore, F.D., Boling, E.A., Ditmore, H.B., Sicular, A., Teterick, J.E., Ellison, A.E., Hoyle, S.J., Ball, M.R., Body Sodium and Potassium V. The Relationship of Alkalosis, Potassium Deficiency and Surgical Stress to Acute Hypokalemia in Man. Metabolism 4:379, 1955.

83. Moore, F.D., Olesen, K.H., McMurrey, J.D., Parker, H.V., Ball, M.R., Boyden, C.M., The Body Cell Mass and its Supporting Environment. Saunders, Philadelphia, 1963, p. 379-406.

84. Ibid, p. 411 - 424.

85. Ibid, p. 431

86. Ibid, p. 519-530.

87. Nahas, G.G., Acid-Base Balance. Anesthesiology 28:787, 1967.

88. Nelson, D.H., Relative Merits of the Adreno-Cortical Steroids. Ann. Rev. Med. 13:241, 1962.

89. Nernst, W., Zur Theorie des elektrischen Reizen, Pflug. Arch des Physiol. 122:275, 1908. as quoted in Cell K., Kernan, R.P., Butterworths, Washington, 1965, p. 59.

90. Olesen, K.H., Parker, H.V., Moore, F.D., Alterations in Body Composition with Preparations of Cardiac Patients for Surgery. Surg. Forum VIII:39, 1957.

91. Orloff, J., Handler, J.S., The Cellular Mode of Action of Antidiuretic Hormone. Amer. J. Med. 36:686, 1964.

92. Paneth, M.R., Sellers, R., Gott, V.L., Weirich, W.L., Allen, P., Read, R.C., Lillehei, C.W., Physiological Studies upon Prolonged Cardiopulmonary Bypass with the Pump Oxygenator with Particular Reference to 1) Acid base balance 2) Siphon caval drainage. J. Thoracic Surg.34:570, 1957.

93. Peirce, E.C., Dabbs, C.H., Metabolic Acidosis and Oxygen Consumption before, during and following total Cardiopulmonary Bypass with and without Hypothermia. Surg. Forum X:597, 1960.

94. Price, H.L., Linde, H.W., Morse, H.T., Central Nervous Actions of Halothane Affecting the Systemic Circulation. Anesthesiology 24:770, 1963.

95. Rabbino, M.D., Dreifus, L.S., Likoff, W., Cardiac Arrhythmias following Intracardiac Surgery. Amer. J. Cardiol. 7:681, 1961.

96. Renold, A.E., Jenkins, D., Forsham, P.H., Thorn, G.W., The Use of Intravenous ACTH on a Study in Quantitative Adrenocortical Stimulation. J. Clin. Endocrin. Metab. 12:763, 1952.

97. Repke, K., Metabolism of Cardiac Glycosides, in New Aspects of Cardiac Glycosides, ed. Wilbrandt, W., vol. 3, Proceeding the First International Pharmacological Meeting Stockholm, 1961, Pergamon, Press London, 1963, p. 47.

98. Rothballer, A.B., The Effects of Catecholamines on the Central Nervous System. Pharmacol. Rev. 11:494, 1959.

99. Royce, P.C., Sayers, G., Purification of Hypothalmic Corticotrophin Releasing Factor. Proc. Soc. exp. Biol. Med. 103:447, 1960.

100. Royce, P.C., Sayers, G., Blood ACTH: Effects of Ether, Pentobarbitol, Epinephrine, and Pain, Endocrinology 63:794, 1958.

101. Schweizer, O., Horland, W.S., Potassium Levels, Acid-Base Balance, and Massive Blood Replacement. Anesthesiology 23:735, 1962.

102. Shivagb, W.A., Moran, W.H., Zimmermann, B., Studies of the Mechanism of Antidiuretic Hormone Secretion and the Post-Commissurotomy Dilutional Syndrome. Ann. Surg. 162:690, 1965.

103. Siggaard Andersen, O., Blood Acid-Base Alignment Nomogram. Scand. J. Clin. & Lab. Invest. 15:211, 1963.

104. Simmons, D.H., Avedon, M., Acid-Base Alterations and Plasma Potassium Concentrations. Amer. J. Physiol. 197:319, 1959.

105. Staffurth, J.S., The Total Exchangeable Potassium in Patients with Hypokalemia. Postgrad. Med. J. 40:4, 1964.

106. Strumia, M.M., Methods of Blood Preservation in General and Preparation, Preservation and Use of Red Cell Suspension. Am. J. Clin. Path.24:260, 1954.

107. Sutherland, E.W., Rall, T.W., The Relation of Adenosine-3', 5' Phosphate and Phosphorylase to the Action of Catecholamines and other Hormones. Pharmacol. Rev. 12:265, 1960.

108. Talso, P.J., Spafford, N., Blaw, M., The Metabolism of Water and Electrolytes in Congestive Heart Failure, II The distribution of water and electrolytes in skeletal muscle in edematous patients with congestive heart failure before and after treatment. J. Clin. and Lab. Med. 41:405, 1953.

109. Tobian, L., Sodium Renal Arterial Distension and the Juxtaglomerular Apparatus. Canad. Med. Ass. J. 90:160, 1964.

110. Van Brunt, E.E., Ganong, W.F., The Effects of Preanesthetic Medication, Anesthesia and Hypothermia on the Endocrine Response to Injury. Anesthiology 24:500, 1963.

111. Vander, A.J., Malvin, R.L., Wilde, W.S., Lapides, J., Sullivan, L.P., McMurray, V.M., Effects of Adrenalectomy and Aldosterone on Proximal and Distal Tubular Sodium Reabsorption. Proc. Soc. exp. Biol. Med. 99:323, 1958.

112. Verney, E.B., The Antidiuretic Hormone and the Factors which Determine its Release. Proc. Roy Soc.135:25, 1947.

113. Virtue, R.W., LeVine, D.S., Aikawa, J.K., Fluid Shifts during the Surgical Period: RISA and S^{35} Determinations Following Glucose, Saline or Lactate Infusion. Ann. Surg. 163:523, 1966.

114. Walker, W.F., Morgan, H.G., Breckenridge, I.M., Watt, A., Ogilvie, R.R., Douglas, D.M., The Biochemical Response to Open Heart Surgery Using Extracorporeal Circulation. Scot. Med. J. 8:141, 1963.

115. Walker, W.G., Wilde, W.S., kinetics of Radiopotassium in the Circulation. Amer. J. Physiol. 170:401, 1952.

116. Weber, G., Singhal, R.L., Stamm, N.B., Actinomycin: Inhibition of Cortisone induced Synthesis of Hepatic Gluconeogenic Enzymes. Science 142:390, 1963.

117. Whittam, R., The Asymmetrical Stimulation of a Membrane Adenosine Triphosphatase in Relation to Active Cation Transport. Biochem. J. 84:110, 1962.

118. Wood, G.A., Levitt, S.H., Simultaneous Red Cell Mass and Plasma Volume Determinations using ^{51}Cr tagged red cells and ^{125}I labelled albumin. J. Nuel. Med. 6:433, 1965.

APPENDIX

APPENDIX 1

MASTER POTASSIUM DIET SHEET

BREAKFAST	K.	Cals.	K.	Cals.	K.	Cals.
Apple, grapefruit, pineapple or grape juice (Limit to ½c/day)	140	50	140	50	140	50
S.F. whole grain cereal(½c) /c sugar (5 gm)	124	70 40	124	70 40	124	70 40
1 egg	50	73	50	73	50	73
S.F. butter - 2 tsp.(1 pat)	-	90(3 tsp)-		135(3 tsp.)		130
S.F. toast - 1 sl.(whole grain)	97	68(1½ sl)	145	102(1½ sl)	145	102
½ cup skim milk	168	40	168	40	168	76
Honey, jam, jelly or marmalade	3	80	3	80	3	80
Instant coffee (1 tsp.= 1 gm.slightly rounded)	32.5	-	32.5	-	32.5	-
Sugar - (5 gm.= 1 lump)		20		20		20
Cream (coffee)	20	20	20	20	20	20
Pepper (no salt)						
	634.5	551	682.5	630	682.5	656

MASTER POTASSIUM DIET SHEET

DINNER	K.	Cals.	K.	Cals.	K.	Cals.
Meat (3 oz.)	211	219	211	219	(4oz) 284	292
Vegetable ($\frac{1}{2}$ cup)	120	40	120	40	120	40
			1 small potato(80 gm)		228	68
			/c butter(1 pat)		-	90
Canned or cooked fruit($\frac{1}{2}$cup)	85 (75)	70	85	70	85	70
S.F. whole wheat bread,1 sl.	97	68	(2) 194 sl)	136	(2sl) 194	136
S.F. butter, 1 pat(2 tsp)	-	90	(3 - tsp)	135	(3 - tsp)	135
Tea	17	-	17	-	17	-
$\frac{1}{2}$ c.(4 oz) skim milk	168	40	168	40	168	80
	698	527	795	640	1096	911

SUPPER						
Same as dinner	698	527	795	640	1096	911

MASTER POTASSIUM DIET SHEET

NOURISHMENT	K.	Cals.	K.	Cals.	K.	Cals.
(May be taken any time during the day)						
½ cup orange juice						
or						
½ of a 6" banana	-	-	180	40	180	40

****

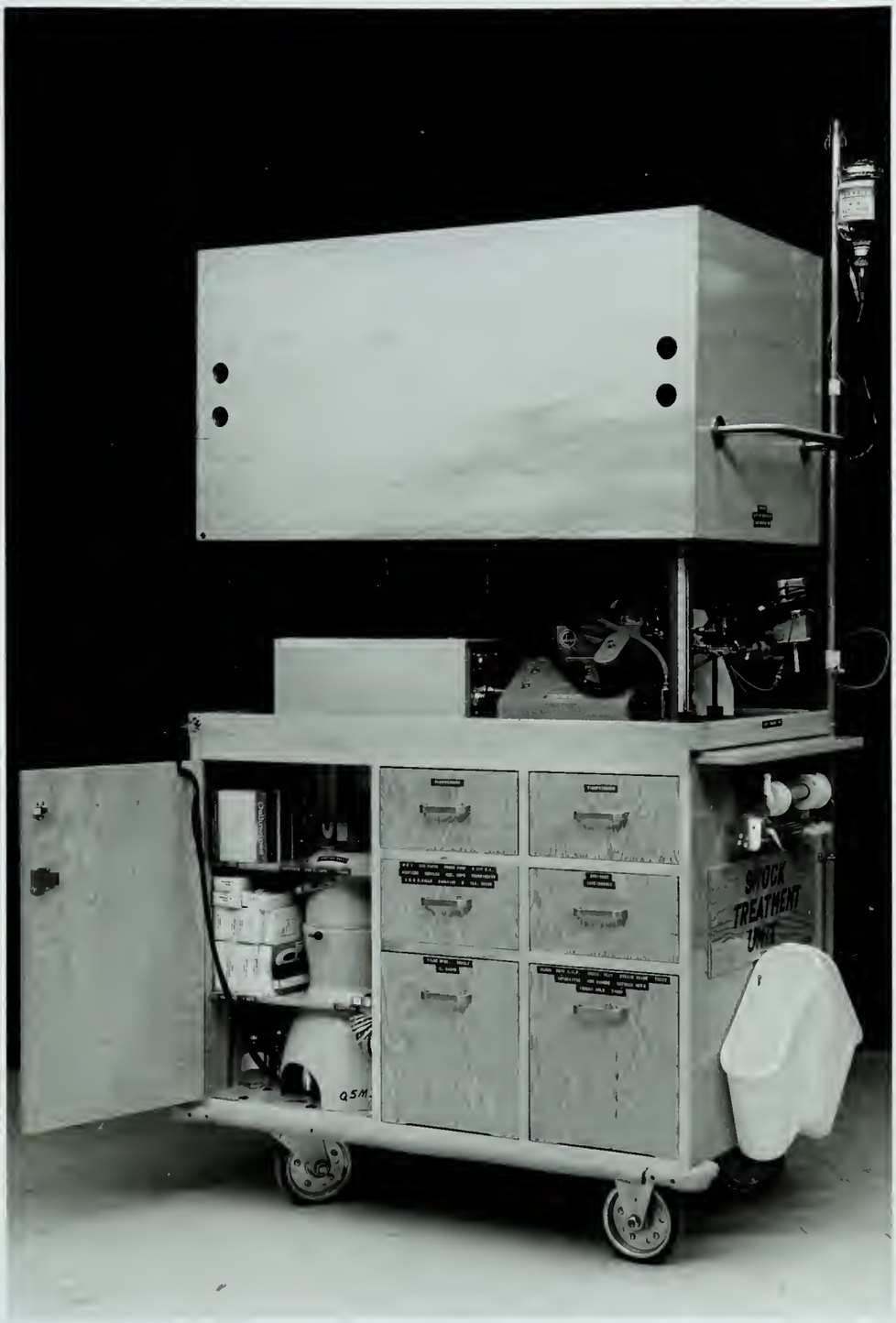
TOTAL - (Recap.)	634.5	551	682.5	630	682.5	656
	698	527	795	640	1096	911
	698	527	795	640	1096	911
			180	40	180	40
	2030.5 mg.	1605	2452.5 mg.	1950	3054.5 mg.	2518
	(1.92-2.24g.)	(1600 Cal.)	(2.4-2.8g.)	(2000 Cal)	(2.88-3.36g)	(2400 Cal)

NOTE:

The above meal patterns need not be followed precisely as outlined as long as the KIND OF FOOD and the AMOUNTS indicated are not varied over the whole day.

APPENDIX 2

BLOOD VOLUME EQUIPMENT


SHOCK
TREATMENT
UNIT

APPENDIX 3

NO. :_____

DATE:_____

BLOOD VOLUME CALCULATION SHEET

Name: Last Hct:

Doctor: Weight at Present:

Ward: Weight on Admission:

Hospital No.:

(All counts represent 2 ml. of solution counted for 5 minutes.)

(Background counts are subtracted automatically.)

1. Patients hct. (micro) =
2. (1 - hct.) = c.p.m.
3. I-125 counts injected = counts in 2 cc. of diluted std. x 20 = c.p.m.
4. Cr-51 counts injected = counts in 2 cc. of ACD solution = c.p.m.
5. Cr-51 counts in empty syringe = counts/5 = c.p.m.
6. I-125 counts in empty syringe = counts/5 = c.p.m.

Sample (S_1) Equilibration Time = _____ Min.

7. I-125 S_1 counts/10 = c.p.m./cc of whole blood
8. Cr-51 S_1 counts/10 = c.p.m./cc of whole blood

CALCULATIONS

9. P.V. = $\frac{\text{I-125 counts injected}}{\text{I-125 } S_1 \text{ counts x } 100/(1\text{-hct.})}$

$= \frac{(3 - 6)}{7 \times 100/(1 - \text{hct.})}$ = cc.

10. R.C.V. = $\frac{\text{Cr-51 counts injected}}{\text{Cr-51 } S_1 \text{ counts x } 100/\text{hct.}}$

$= \frac{(4 - 5)}{8 \times 100/\text{hct.}}$ = cc.

11. T.B.V. = P.V. + R.C.V. = 9 + 10 = cc.

12. Wt. in Kg. = lbs./2.2 = Kg.

13. P.V. in cc/Kg. = 9/12 = cc/Kg.

14. R.C.V. in cc/Kg. = 10/12 = cc/Kg.

15. T.B.V. in cc/Kg. = 11/12 = cc/Kg.

16. Whole Body Hematocrit = $\frac{\text{R.C.V.}}{\text{P.V. + R.C.V.}}$

$= \frac{10}{9 + 10}$ =

Miscellaneous

S_2 - Cr-51 counts = S_3 - Cr =

I-125 counts = I =

S_4 - Cr = S_5 - Cr =

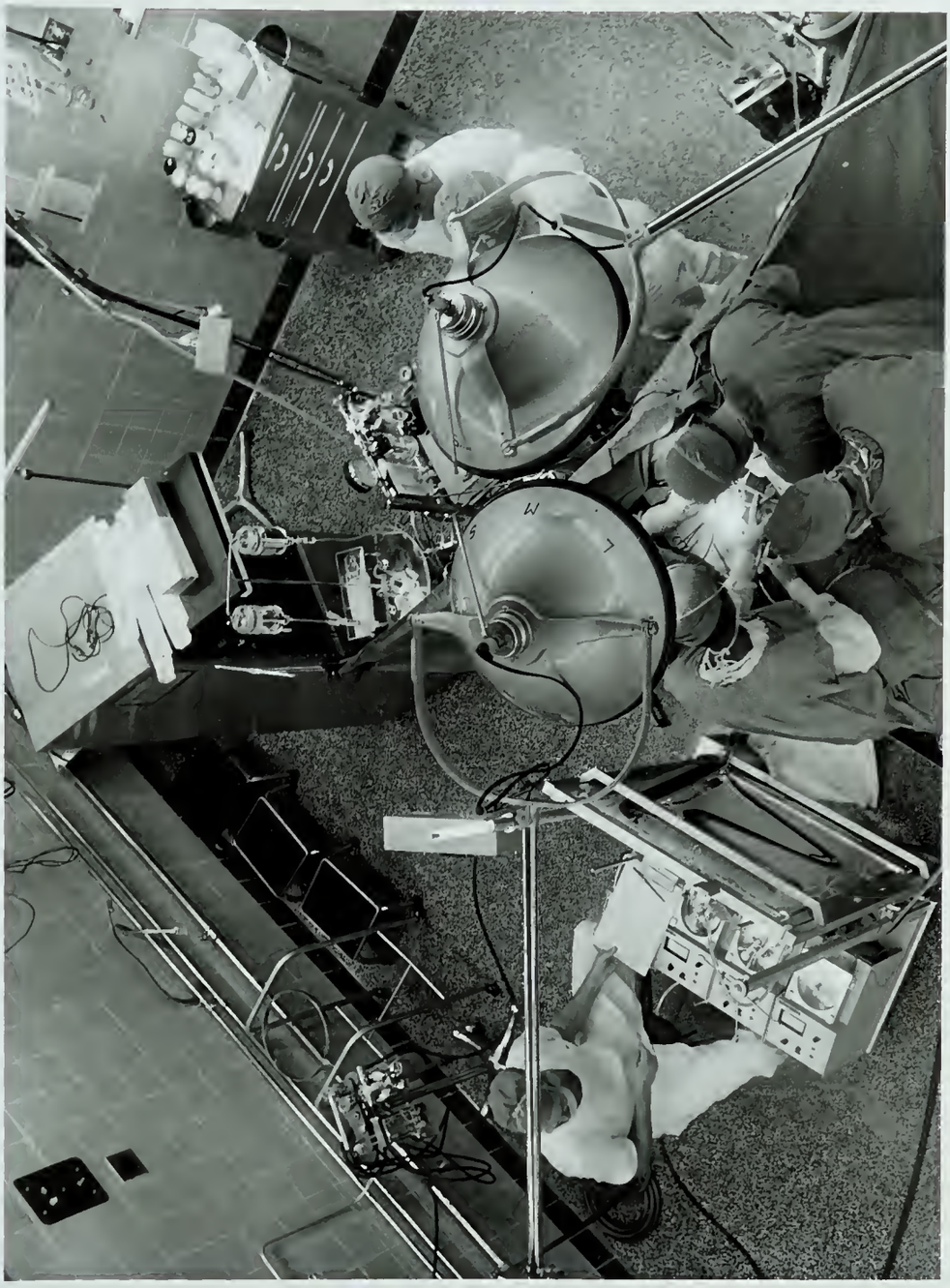
I = I =

BLOOD VOLUME RESEARCH

1. Time allowed for tagging = min.
2. ACD bottle Hct. =
3. (1 - ACD Hct.) =
4. Extrapolation of I cts. to zero from S_1, S_2 and S_3 = counts
5. Extrapolation of Cr cts. to zero from S_1, S_2 and S_3 = counts
6. Cr cts in 2cc of ACD plasma = counts
7. Total unbound (plasma) Cr injected
   = 6 x 3/100 = counts
8. % of Cr cts in plasma
   $$= \frac{7 \times 100}{\text{Net Cr counts injected}}$$ = %
9. Total tagged Cr injected
   = Net Cr injected - 7 = counts
10. Cr overlap in I window = .012 x net Cr cts. = counts
11. Recalculated R.C.V. $= \frac{9}{5 - \text{Cr counts in } S_1 \text{ plasma}}$ = cc.
12. Recalculated P.V. $= \frac{\text{Total I counts injected}}{4 - 10}$ = cc.
13. Recalculated T.B.V. = 11 + 12 = cc.
14. Plasma Cr dilution (if available) =
15. Actual Whole Body Hematocrit $= \frac{11}{11 + 12}$ =

APPENDIX 4

CARDIOPULMONARY SURGERY DURING BYPASS



APPENDIX 5

OXYGEN CONSUMPTION

Sample Calculation

Case #8

Height = 5' 6"

Weight = 68 Kg.

Surface Area (cm^2) (42) = $W(Kg.)^{0.425} \times H(cm)^{0.725} \times 71.84$

= $(68)^{0.425} \times (68)(2.54)^{0.725} \times 71.84$

Blood Flow = $\frac{\text{Oxygen uptake} \times 100}{\text{arterial - venous oxygen difference}}$

Arterial-venous difference = (arterial saturation-venous saturation)Hb.

+ 0.003 x (arterial tension-venous tension

Haemoglobin = 9.1

Oxygen uptake x 100 = 2800 x ((.99 -.59)(15.8)(27) + .003 x (230 - 30))

= 2800 x ((.40)(90.8) + .6)

= 2800 (3.63 + .6)

= 2800 x 4.23

= 11790

Oxygen uptake = $\frac{11790}{100}$

= 117.0 cc/min.

Oxygen uptake/minute/ m^2 body surface area = $\frac{117.9}{S.A.}$

= $\frac{117.9}{\frac{(68)^{0.425} \times (68)(2.54)^{0.725} \times 71.84}{100 \times 100^*}}$

$$= \frac{117.9}{1.55}$$

$$= 76 \text{ ml./min./m}^2$$

* converts cm^2 to m^2

APPENDIX 6

INDIVIDUAL CASE RECORDS
OF
TWENTY-SIX
CARDIOPULMONARY OPERATIONS

Name - R.M.
Case No - 1
Age - 46
Sex - M

Ht- 67"
Wt- 152 lbs.
- 69 Kg.
Body Surface Area (m^2) -

Operation - Aortic Valve Replacement

Perf.Time - 98 mins
Group - II

Meds. - nil

Arrhythmias - nil

Hypothermia - no

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.0	4.2	4.7	3.9	4.5	4.7	4.7	5.0	4.2
Gases	art.pO_2	(mmHg)		186	200	400	316	320	220		
	ven.pO_2	(mmHg)									
	art.pCO_2	(mmHg)		32	36	39	37	26	32		
	ven.pCO_2	(mmHg)									
	pH			7.55	7.45	7.42	7.47	7.52	7.45		
	art.HCO_3	(mEq/l)		28	25	24	26	22	22		
	act.HCO_3	(mEq/l)		27	24	24	26	20	21		
	art.Sat.	(%)		99	99	99	99	99	99		
	ven.Sat.	(%)									
Flow		(cc)			2200	3200	3100	3000			
		(cc/Kg)			32	46	45	43			
O_2Cons.(ml/min/m^2BSA)											
Hematocrit			45	42	29	28	28	30	41	36	33
Hemoglobin		(gm%)	14.6	13.6	9.4	9.1	9.1	9.7	13.4	11.7	10.6
Lactate & Saline		(cc)				2600					
Urine Volume		(cc)		50		42			180	520	
Urine Potassium		(mEq)		3.9		2.7			9.0		
Net Fluid Change		(cc)								2265	
Red Cell Volume		(cc)	1770							1810	
Plasma Volume		(cc)	2820							3200	

Name - W.H. Ht-75"
Case No - 2 Wt-183 lbs.
Age - 42 -83 Kg.
Sex - M Body Surface Area (m^2) -

Operation - Mitral Valve Replacement

Perf.Time - 98 mins.
Group - II

Meds. - Digitalis (pre-op) (post-op)
Arrhythmias - Atrial Fibrillation (post-op)
Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	3.4	4.8	4.1	4.3	4.4	4.7	4.3	4.9	4.0
Gases	art.pO_2	(mmHg)		186	116	235	280	270	106		
	ven.pO_2	(mmHg)									
	art.pCO_2	(mmHg)		43	38	32	35	34	34		
	ven.pCO_2	(mmHg)									
	pH			7.42	7.41	7.50	7.47	7.48	7.40		
	art.HCO_3	(mEq/l)		26	23	25	26	26	22		
	act.HCO_3	(mEq/l)		27	23	24	25	24	20		
	art.Sat.	(%)		99	99	99	99	99	99		
	ven.Sat.	(%)									
Flow		(cc)			3800	3800	3800	3800			
		(cc/Kg)			46	46	46	46			
O_2Cons.(ml/min/m^2BSA)											
Hematocrit			47	45	29	31	31	29	35	43	
Hemoglobin		(gm%)	16.6	15.9	10.2	10.9	10.9	10.2	12.4	15.2	
Lactate & Saline		(cc)				2450					
Urine Volume		(cc)		50		42			180	820	
Urine Potassium		(mEq)		5.0		2.7			2.5		
Net Fluid Change		(cc)								1924	
Red Cell Volume		(cc)	1980							1670	
Plasma Volume		(cc)	2603							2840	

Name - F.W. Ht- 62"
Case No - 3 Wt- 127 lbs.
Age - 49 - 58 Kg.
Sex - F Body Surface Area (m^2) -

Operation - Mitral Valve Replacement & Tricuspid annuloplasty

Perf.Time - 155 mins.
Group - II

Meds. - Digitalis (pre-op)
(post-op)
Arrhythmias - nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	3.1	4.5	3.7	4.0	4.3	4.9	4.8	3.5	3.8
Gases	art.pO_2	(mmHg)		66	160	214	234	170	174		
	ven.pO_2	(mmHg)									
	art.pCO_2	(mmHg)		57	37	37	40	37	31		
	ven.pCO_2	(mmHg)									
	pH			7.38	7.43	7.45	7.44	7.46	7.52		
	art.HCO_3	(mEq/l)		26	24	25	26	26	26		
	act.HCO_3	(mEq/l)		27	25	26	28	26	24		
	art.Sat.	(%)		91	99	99	99	99	99		
	ven.Sat.	(%)									
Flow		(cc)			2600	2500	2500	2600			
		(cc/Kg)			45	43	43	45			
O_2Cons.(ml/min/m^2BSA)											
Hematocrit			41	36	28	31	31	31	35	43	41
Hemoglobin		(gm%)	13.6	11.9	9.3	10.3	10.3	10.3	11.6	13.1	12.4
Lactate & Saline		(cc)				2100					
Urine Volume		(cc)		53		50			38	1100	
Urine Potassium		(mEq)		4.1		3.1			2.8	110	
Net Fluid Change		(cc)								1556	
Red Cell Volume		(cc)									
Plasma Volume		(cc)									

Name - A.W. Ht- 62"
Case No - 4 Wt- 135 lbs.
Age - 55 - 61 Kg.
Sex - F Body Surface Area (m^2) -

Operation - Aortic Valve Replacement
Perf.Time - 76 mins.
Group - II

Meds. - Diuretics
Digitalis (pre-op, post-op)
Arrhythmias - nil

Hypothermia No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	3.9	5.3	3.5	3.7	4.3	4.5	4.9	4.7	3.3	3.7
Gases	art.pO_2	(mmHg)		75	302	340	360	390	144			
	ven.pO_2	(mmHg)										
	art.pCO_2	(mmHg)		28	34	36	37	30	25			
	ven.pCO_2	(mmHg)										
	pH			7.51	7.44	7.42	7.46	7.49	7.57			
	art.HCO_3	(mEq/l)		24	23	23	26	25	25			
	act.HCO_3	(mEq/l)		23	23	24	26	22	22			
	art.Sat.	(%)		99	99	99	99	99	99			
	ven.Sat.	(%)										
Flow		(cc)			2800	3100	2600	2600				
		(cc/Kg)			45	50	42	42				
O_2Cons.(ml/min/m^2BSA)												
Hematocrit			42	33	23	25	26	24	26		38	
Hemoglobin		(gm%)	14.2	11.2	7.8	8.5	8.8	8.2	8.8		12.9	
Lactate & Saline		(cc)				1700						
Urine Volume		(cc)		0		80			40		550	
Urine Potassium		(mEq)		0.0		4.5			1.2		29	
Net Fluid Change		(cc)									1960	
Red Cell Volume		(cc)										
Plasma Volume		(cc)										

Name - A.D.
Case No - 5
Age - 51
Sex - F

Ht- 64"
Wt- 107 lbs.
- 49 Kg.
Body Surface Area (m^2) -

Operation - Aortic Valve Replacement

Perf.Time - 98 mins.
Group - II

Meds. - Diuretics; Digitalis (pre-op, post-op)
Arrhythmias - nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K	(mEq/l)	4.1	4.4	4.0	3.8	3.8	4.1	3.9	4.3	3.4	3.1
	art.pO_2	(mmHg)		109	105	95	205	330	118			
	ven.pO_2	(mmHg)										
	art.pCO_2	(mmHg)		27	39	36	43	34	38			
	ven.pCO_2	(mmHg)										
	pH			7.45	7.33	7.36	7.33	7.37	7.40			
	art.HCO_3	(mEq/l)		22	21	20	23	20	23			
	act.HCO_3	(mEq/l)		19	18	20	23	19	23			
	art.Sat.	(%)		99	99	99	99	99	99			
	ven.Sat.	(%)										
Flow		(cc)			2100	2500	2500	2500				
		(cc/Kg)			43	51	51	51				
O_2Cons.(ml/min/m^2BSA)												
Hematocrit			39	34	25	26	30	25	31	40	42	
Hemoglobin		(gm%)	13.6	11.8	8.7	9.1	10.4	8.7	10.8	13.9	14.6	
Lactate & Saline		(cc)				1650						
Urine Volume		(cc)		20		95			40	890		
Urine Potassium		(mEq)		2.5		5.9			2.8	73		
Net Fluid Change		(cc)								1445		
Red Cell Volume		(cc)	1144							1138		
Plasma Volume		(cc)	2088							2067		

Name - E.N.
Case No - 6
Age - 57
Sex - F

Ht- 62"
Wt- 105 lbs.
- 48 Kg.
Body Surface Area (m^2) -

Operation -Aortic Valve Replacement & Mitral Valvotomy

Perf.Time - 122 mins.
Group - II

Meds. - Nil

Arrhythmias- Atrial Tachycardia & Atrial Fibrillation on Bypass
Hypothermia - No

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K (mEq/l)	3.8	5.2	4.6	4.6	5.6	5.7	4.1	died day #1	
	art.pO_2 (mmHg)		75	280	295	323	480	340		
	ven.pO_2 (mmHg)									
	art.pCO_2 (mmHg)		43	37	31	26	28	33		
	ven.pCO_2 (mmHg)									
	pH		7.31	7.36	7.42	7.49	7.44	7.38		
	art.HCO_3 (mEq/l)		21	21	22	21	20	20		
	act.HCO_3 (mEq/l)		21	20	22	21	18	19		
	art.Sat. (%)		93	99	99	99	99	99		
	ven.Sat. (%)									
Flow	(cc)			2500	2600	2600	1900			
	(cc/Kg)			52	54	54	40			
O_2Cons.(ml/min/m^2BSA)										
Hematocrit		41	39	21	21	25	31	25		
Hemoglobin	(gm%)	13.1	12.5	6.7	6.7	8.0	9.9	8.0		
Lactate & Saline	(cc)				2000					
Urine Volume	(cc)		45		130			28		
Urine Potassium	(mEq)		2.1		3.4			0.8		
Net Fluid Change	(cc)								1997	
Red Cell Volume	(cc)									
Plasma Volume	(cc)									

Name - J.W. Ht- 67"
Case No - 7 Wt- 156 lbs.
Age - 53 - 71 Kg.
Sex - M Body Surface Area (m^2) -

Operation - Mitral Valve Replacement

Perf.Time - 141 mins.
Group - I II

Meds. - Digitalis (post-op)

Arrhythmias - nil

Hypothermia - 31°C

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K (mEq/l)	3.9	6.0	5.8	5.5	4.7	4.3	3.9	6.5	died day #2
	art.pO_2 (mmHg)		116	120	280	70	83	196		
	ven.pO_2 (mmHg)									
	art.pCO_2 (mmHg)		46	33	27	29	29	38		
	ven.pCO_2 (mmHg)									
	pH		7.59	7.48	7.52	7.53	7.51	7.55		
	art.HCO_3 (mEq/l)		26	23	24	25	24	25		
	act.HCO_3 (mEq/l)		23	22	24	25	23	24		
	art.Sat. (%)		99	99	99	99	99	96		
	ven.Sat. (%)									
Flow	(cc)			4000	4000	4000	4000			
	(cc/Kg)			57	57	57	57			
O_2Cons.(ml/min/m^2BSA)										
Hematocrit		43	44	29	33	34	31	44	38	
Hemoglobin	(gm%)	15.3	15.6	10.3	11.7	12.1	10.0	15.6	13.5	
Lactate & Saline	(cc)				2800					
Urine Volume	(cc)							(300)	Nil	
Urine Potassium	(mEq)				no catheter			(13.0)		
Net Fluid Change	(cc)								2600	
Red Cell Volume	(cc)	1920							1890	
Plasma Volume	(cc)	3034							3670	

Name - A.L.
Case No - 8
Age - 60
Sex - M

Ht- 66"
Wt- 150 lbs.
- 68 Kg.
Body Surface Area (m^2) - $1.82M^2$

Operation - Aortic Valve Replacement

Perf.Time - 97 mins.
Group - II

Meds. - Diabetic on Tolbutamide; Digitalis (post-op)
Arrhythmias - nil

Hypothermia - No

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K (mEq/l)	4.6	5.7	5.7	5.5	5.5	6.0	5.7	3.9	4.2
Gases	art.pO_2 (mmHg)		160	230	295	230	380	170		
	ven.pO_2 (mmHg)		44	30	27	43	46	52		
	art.pCO_2 (mmHg)		40	24	24	32	22	28		
	ven.pCO_2 (mmHg)		43	32.5	30.5	41	36	43		
	pH		7.40	7.51	7.52	7.44	7.51	7.34		
	art.HCO_3 (mEq/l)		26	21	22	22	20	20		
	act.HCO_3 (mEq/l)		26	18	19	21	17	20		
	art.Sat. (%)		99	99	99	99	99	99		
	ven.Sat. (%)		73	59	54	66	73	73		
Flow	(cc)			2800	2600	3000	3000			
	(cc/Kg)			41	38	44	44			
O_2Cons.(ml/min/m^2BSA)				65	66	64	56			
Hematocrit		47	42	27	25	25	26	31	45	
Hemoglobin	(gm%)	15.8	14.1	9.1	8.4	8.4	8.7	10.4	15.1	
Lactate & Saline	(cc)				2500					
Urine Volume	(cc)		45		90			130	1936	
Urine Potassium	(mEq)		8.0		6.6			8.2	139	
Net Fluid Change	(cc)								1485	
Red Cell Volume	(cc)	1583							1339	
Plasma Volume	(cc)	2243							1685	

Name - J.P.
Case No - 9
Age - 52
Sex - M

Ht- 68"
Wt- 139 lbs.
- 63 Kg.
Body Surface Area (m^2) -1.78 M^2

Operation - Aortic Valve Replacement

Perf.Time - 95 mins.
Group - II

Meds. - Diuretics; Digitalis (pre-op, post-op)
Arrhythmias- nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	3.2	4.2	4.1	4.3	4.4	4.5	3.3	4.0	3.8
Gases	art.pO_2	(mmHg)		142	260	380	410	430	182		
	ven.pO_2	(mmHg)		25	40	32	34	38	35		
	art.pCO_2	(mmHg)		40	34	29	31	31.5	45.5		
	ven.pCO_2	(mmHg)		45	38.5	33.5	36.5	38	49		
	pH			7.42	7.52	7.55	7.56	7.59	7.48		
	art.HCO_3	(mEq/l)		27	25	29	28	28	23		
	act.HCO_3	(mEq/l)		27	24	28	26	23	27		
	art.Sat.	(%)		99	99	99	99	99	99		
	ven.Sat.	(%)		48	77	76	77	77	74		
Flow		(cc)			3100	2600	2500	2500			
		(cc/Kg)			50	41	40	40			
O_2Cons.(ml/min/m^2BSA)					45	51	53	48			
Hematocrit			46	47	28	33	32	35	41		
Hemoglobin		(gm%)	14.4	14.7	8.8	10.3	10.0	10.9	12.8		
Lactate & Saline		(cc)				2300					
Urine Volume		(cc)		40		200			210	570	
Urine Potassium		(mEq)		2.3		8.2			13.4	43.8	
Net Fluid Change		(cc)								1085	
Red Cell Volume		(cc)	1835							1305	
Plasma Volume		(cc)	2790							2545	

Name - V.S.
Case No - 10
Age - 44
Sex - F

Ht- 60"
Wt- 107 lbs.
- 48 Kg.
Body Surface Area (m^2) -1.47 M^2

Operation -Mitral Valve & Tricuspid Replacements

Perf.Time - 148 mins.
Group - II I

Meds. Digitalis (pre-op)

Arrhythmias- nil

Hypothermia - 31° C

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.1	5.0	4.6	4.6	4.6	5.0	4.9	4.0	4.0
Gases	art.pO_2	(mmHg)		70	112	194	245	300	98		
	ven.pO_2	(mmHg)		30	28	30	35	40	28		
	art.pCO_2	(mmHg)		26	39	31	33	31	35		
	ven.pCO_2	(mmHg)		29	43	31.5	37	35	42		
	pH			7.51	7.35	7.39	7.42	7.44	7.35		
	art.HCO_3	(mEq/l)		23	21	20	22	22	20		
	act.HCO_3	(mEq/l)		21	23	20	21	22	19		
	art.Sat.	(%)		95	99	99	99	99	98		
	ven.Sat.	(%)		61	52	61	68	71	47		
Flow		(cc)			2600	2500	2300	2500			
		(cc/Kg)			54	52	48	52			
O_2Cons.(ml/min/m^2BSA)					103	65	52	69			
Hematocrit			52	48	39	37	35	36	39	39	
Hemoglobin		(gm%)	15.9	14.6	11.9	11.3	10.7	11.0	11.9	11.8	
Lactate & Saline		(cc)				1900					
Urine Volume		(cc)		11		170			22	590	
Urine Potassium		(mEq)		0.7		9.8			1.0	77	
Net Fluid Change		(cc)								1037	
Red Cell Volume		(cc)									
Plasma Volume		(cc)									

Name - D.K.
Case No - 11
Age - 52
Sex - M

Ht- 66"
Wt- 159 lbs.
- 72 Kg.
Body Surface Area (m^2) - 1.85 M^2

Operation - Mitral Valve Replacement

Perf.Time - 76 mins.
Group - II

Meds.- Diuretics - Potassium chloride, Digitalis (pre-op)
Arrhythmias- nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	3.3	4.3	5.6	5.5	5.8	6.3	4.6	4.2	4.1	2.8
Gases	art.pO_2	(mmHg)		110	66	138	310	490	134			
	ven.pO_2	(mmHg)			30	34	36	50	43			
	art.pCO_2	(mmHg)		31	33	39	40	21	34			
	ven.pCO_2	(mmHg)			33	39	41.5	35.5	40			
	pH			7.40	7.47	7.44	7.45	7.67	7.44			
	art.HCO_3	(mEq/l)		23	24	26	28	28	24			
	act.HCO_3	(mEq/l)		22	23	26	28	23	22			
	art.Sat.	(%)		96	92	99	99	99	99			
	ven.Sat.	(%)			62	68	71	87	77			
Flow		(cc)			3000	3000	3000	3000				
		(cc/Kg)			41	41	41	41				
O_2Cons.(ml/min/m^2BSA)					60	71	72	46				
Hematocrit			58	52	35	38	36	34	41		38	
Hemoglobin		(gm%)	19.8	17.7	12.0	13.0	12.3	11.6	14.0		12.0	
Lactate & Saline		(cc)				2140						
Urine Volume		(cc)		20		90			110		750	
Urine Potassium		(mEq)		0.8		6.0			4.0		74	
Net Fluid Change		(cc)									1656	
Red Cell Volume		(cc)	1930								1925	
Plasma Volume		(cc)	2505								2780	

Name - G.D.
Case No - 12
Age - 45
Sex - M

Ht-73"
Wt-179 lbs.
-81 Kg.
Body Surface Area (m^2) -2.06 M^2

Operation - Aortic Valve Replacement

Perf.Time - 112 mins.
Group - II

Meds. - Nil

Arrhythmias - Ventricular Fibrillation During Bypass
Hypothermia - 31°C

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K (mEq/l)	3.6	5.0	4.9	4.3	5.2	5.4	4.2	4.6	3.2
Gases	art.pO_2 (mmHg)		102	149	255	349	460	75		
	ven.pO_2 (mmHg)		38	27	33	42	38	47		
	art.pCO_2 (mmHg)		33	38	36	38	24	33		
	ven.pCO_2 (mmHg)		34.5	28.5	37	41	40	31		
	pH		7.47	7.41	7.43	7.45	7.61	7.46		
	art.HCO_3 (mEq/l)		24	24	24	26	26	23		
	act.HCO_3 (mEq/l)		24	23	23	26	23	23		
	art.Sat. (%)		98	99	100	100	100	94		
	ven.Sat. (%)		78	56	77	76	77	82		
Flow	(cc)			1800	2000	3300	2900			
	(cc/Kg)			22	25	41	36			
O_2Cons.(ml/min/m^2BSA)				44	55	60	57			
Hematocrit		48	46	30	34	35	34	36	43	
Hemoglobin	(gm%)	16.5	15.8	10.3	11.7	12	11.7	12.4	14.7	
Lactate & Saline	(cc)				3300					
Urine Volume	(cc)							(300)	2390	
Urine Potassium	(mEq)				no catheter			(16.9)	143	
Net Fluid Change	(cc)								3522	
Red Cell Volume	(cc)	1890							2200	
Plasma Volume	(cc)	3165							3800	

Name - W.R.
Case No - 13
Age - 42
Sex - M

Ht- 74"
Wt- 195 lbs.
- 81 Kg.
Body Surface Area (m^2) - 2.14 M^2

Operation -Aortic Valve & Aortic Arch Replacements

Perf.Time - 150 mins.
Group - III

Meds.- Digitalis (post-op)

Arrhythmias- nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.2	6.2	5.7	5.5	5.6	5.5	5.1	4.2	4.1
Gases	art.pO_2	(mmHg)		58	270	280	310	340	300		
	ven.pO_2	(mmHg)		22	36	38	34	40			
	art.pCO_2	(mmHg)		35	23	25	18	22	34		
	ven.pCO_2	(mmHg)		47	26	25.5	25.5	24			
	pH			7.45	7.51	7.49	7.55	7.49	7.40		
	art.HCO_3	(mEq/l)		24	21	20	18	18	21		
	act.HCO_3	(mEq/l)		23	18	18	16	14	20		
	art.Sat.	(%)		90	99	99	99	94	99		
	ven.Sat.	(%)		37	71	77	68	79			
Flow		(cc)			3500	3800	3500	3600			
		(cc/Kg)			40	43	40	41			
O_2Cons.(ml/min/m^2BSA)					55	53	71	82			
Hematocrit			40	41	26	28	31	33	36		
Hemoglobin		(gm%)	14.6	15.0	9.5	10.2	11.3	12.0	13.1		
Lactate & Saline		(cc)				3400					
Urine Volume		(cc)		25		140			65	1920	
Urine Potassium		(mEq)		2.7		13.6			5.5	240	
Net Fluid Change		(cc)								2570	
Red Cell Volume		(cc)	2220							1640	
Plasma Volume		(cc)	3640							3355	

Name - M.S.
Case No - 14
Age - 44
Sex - F

Ht- 67"
Wt- 130 lbs.
- 59 Kg.
Body Surface Area (m^2) - 1.69 M^2

Operation - A.S.D. Repair

Perf.Time - 32 mins.
Group - I

Meds.- Nil

Arrhythmias- nil

Hypothermia - No

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K (mEq/l)	3.8	4.7		4.5		4.7	4.3	4.1	not available, patient discharged
Gases	art.pO_2 (mmHg)		100		63		440	34		
	ven.pO_2 (mmHg)				30		34	35		
	art.pCO_2 (mmHg)		26		25		35	51		
	ven.pCO_2 (mmHg)				27		51	63		
	pH		7.53		7.44		7.41	7.28		
	art.HCO_3 (mEq/l)		24		19		22	22		
	act.HCO_3 (mEq/l)		21		16		21	24		
	art.Sat. (%)		98		85		109	84		
	ven.Sat. (%)				58		75	55		
Flow	(cc)				2500		2700			
	(cc/Kg)				43		47			
O_2Cons.(ml/min/m^2BSA)					44		63			
Hematocrit		43	39		29		33	41	46	
Hemoglobin	(gm%)	14.4	13.0		9.7		11.0	13.7	15.4	
Lactate & Saline	(cc)						1500			
Urine Volume	(cc)								800	
Urine Potassium	(mEq)						no catheter		44	
Net Fluid Change	(cc)								844	
Red Cell Volume	(cc)	1440							1350	
Plasma Volume	(cc)	2672							1850	

Name - G.F. Ht- 72"
Case No - 15 Wt- 163 lbs.
Age - 29 - 74 Kg.
Sex - M Body Surface Area (m^2) - 1.76 M^2

Operation - A.S.D. Repair & Mitral plication
Perf.Time - 119 mins.
Group - II

Meds. - Digitalis (post-op)
Arrhythmias- nil
Hypothermia - 31°C

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass		Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.6	4.4	4.7	4.1	4.3	4.8	4.3	4.0	4.1	3.8
Gases	art.pO_2	(mmHg)		79	86	249	255	410	70			
	ven.pO_2	(mmHg)		40	36	39	44	56	45			
	art.pCO_2	(mmHg)		34	55	32	38	30	43			
	ven.pCO_2	(mmHg)		33	36.5	34.5	45	33.5	55			
	pH			7.40	7.25	7.37	7.34	7.43	7.27			
	art.HCO_3	(mEq/l)		22	19	19	20	21	19			
	act.HCO_3	(mEq/l)		21	20	19	21	19	19			
	art.Sat.	(%)		95	92	99	99	99	90			
	ven.Sat.	(%)		73	64	72	86	88	73			
Flow		(cc)			3200	3300	3600	3700				
		(cc/Kg)			43	45	49	50				
O_2Cons.(ml/min/m^2BSA)					64	58	51	55				
Hematocrit			52	49	36	36	39	38	43		44	
Hemoglobin		(gm%)	18.0	17.0	12.4	12.4	13.5	13.1	14.9		15.2	
Lactate & Saline		(cc)				3300						
Urine Volume		(cc)		15		170			150		925	
Urine Potassium		(mEq)		1.8		8.3			6.9		97	
Net Fluid Change		(cc)									2265	
Red Cell Volume		(cc)	2550								1700	
Plasma Volume		(cc)	3120								2990	

Name - G.H.
Case No - 16
Age - 63
Sex - F

Ht- 64"
Wt- 122 lbs.
- 55 Kg.
Body Surface Area (m^2) - 1.60 M^2

Operation - Mitral Valve Replacement

Perf.Time - 44 mins.
Group - I

Meds.- Digitalis (pre-op, post-op)
Arrhythmias- Atrial Fibrillation During Bypass
Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.2	4.4	5.0	5.0			4.5	4.9	not
Gases	art.pO_2	(mmHg)		95	128	230			180		available
	ven.pO_2	(mmHg)		31	34.5	38					
	art.pCO_2	(mmHg)		35	50	38			34		
	ven.pCO_2	(mmHg)			50	36			39		
	pH			7.36	7.30	7.42			7.38		
	art.HCO_3	(mEq/l)		23	35	23			23		
	act.HCO_3	(mEq/l)		22	23	23			21		
	art.Sat.	(%)		96	99	99			99		
	ven.Sat.	(%)			68	77					
Flow		(cc)			2500	2700					
		(cc/Kg)			45	49					
O_2Cons.(ml/min/m^2BSA)						50	48				
Hematocrit			48	48	26	32			44	47	
Hemoglobin		(gm%)	15.4	15.5	8.3	10.2			14.1	15.0	
Lactate & Saline		(cc)				1400					
Urine Volume		(cc)		20		80			20	580	
Urine Potassium		(mEq)		0.8		4.9			0.8	67	
Net Fluid Change		(cc)								526	
Red Cell Volume		(cc)	1586							1055	
Plasma Volume		(cc)	2025							2227	

Name - E.T.
Case No - 17
Age - 50
Sex - F

Ht- 67"
Wt- 118 lbs.
- 54 Kg.
Body Surface Area (m^2) - 1.63 M^2

Operation - Mitral Annuloplasty

Perf.Time - 20 mins.
Group - I

Meds. - Nil

Arrhythmias- nil

Hypothermia - No

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K (mEq/l)	4.1	4.3	4.4	4.4			4.1	3.7	4.8	3.9
Gases	art.pO_2 (mmHg)		122	63	210			112			
	ven.pO_2 (mmHg)		32	24	37						
	art.pCO_2 (mmHg)		35	30	25			33			
	ven.pCO_2 (mmHg)		38	33.5	30.5			37			
	pH		7.41	7.43	7.51			7.44			
	art.HCO_3 (mEq/l)		22	21	22			22			
	act.HCO_3 (mEq/l)		21	19	19			21			
	art.Sat. (%)		99	92	99			99			
	ven.Sat. (%)		60	44	72			68			
Flow	(cc)			2300	2700						
	(cc/Kg)			43	50						
O_2Cons.(ml/min/m^2BSA)				62	44						
Hematocrit		49	42	27	28			34		41	
Hemoglobin	(gm%)	16.3	14.0	9.0	9.3			11.3		13.6	
Lactate & Saline	(cc)				1600						
Urine Volume	(cc)		0		160			100		540	
Urine Potassium	(mEq)		0.0		8.7			5.2		44	
Net Fluid Change	(cc)									1048	
Red Cell Volume	(cc)	1378								1220	
Plasma Volume	(cc)	1965								2235	

Name - S.M. Ht- 62" Operation - A.S.D. Repair Meds. - Digitalis (post-op)
Case No - 18 Wt- 109 lbs.
Age - 64 - 50 Kg. Arrhythmias - Bigeminal rhythm (post-op), Arrested day #2
Sex - F Body Surface Area (m^2) -1.47 M^2 Perf.Time - 28 mins. Hypothermia - No
Group - I

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1		Post-op Day 5
Blood	K	(mEq/l)	4.1	5.0		4.7		4.9	4.0	4.3	5.1	3.8
Gases	art.pO_2	(mmHg)		72		295		460	110			
	ven.pO_2	(mmHg)		39		52.5		62	50			
	art.pCO_2	(mmHg)		38		58		48	49			
	ven.pCO_2	(mmHg)		40		58		53	62			
	pH			7.46		7.25		7.28	7.30			
	art.HCO_3	(mEq/l)		27		23		21	22			
	act.HCO_3	(mEq/l)		26		24		22	23			
	art.Sat.	(%)		94		99		99	99			
	ven.Sat.	(%)		75		88		87	77			
Flow		(cc)				2500		2600				
		(cc/Kg)				50		52				
O_2Cons.(ml/min/m^2BSA)						28		41				
Hematocrit			37	36		26		24	31			
Hemoglobin		(gm%)	12.5	12.2		8.8		8.1	10.5			
Lactate & Saline		(cc)						1200				
Urine Volume		(cc)		20				15	25		730	
Urine Potassium		(mEq)									73	
Net Fluid Change		(cc)									390	
Red Cell Volume		(cc)										
Plasma Volume		(cc)										

Name - J.W.
Case No - 19
Age - 51
Sex - M

Ht- 64"
Wt- 155 lbs.
- 70 Kg.
Body Surface Area (m^2) - 1.75 M^2

Operation - Aortic Valve Replacement

Perf.Time - 76 mins.
Group - II

Meds.- Digitalis (post-op)

Arrhythmias- nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.1	5.7	5.5	5.1	5.5	5.6	5.0	4.9	4.8	3.9
Gases	art.pO_2	(mmHg)		162	170	345	386	470	99			
	ven.pO_2	(mmHg)		42	35	42	34	31	36			
	art.pCO_2	(mmHg)		44	26	50	26	19	36			
	ven.pCO_2	(mmHg)		41	35	48	40.5	39	45			
	pH			7.50	7.60	7.41	7.51	7.62	7.37			
	art.HCO_3	(mEq/l)		33	27	30	22	22	21			
	act.HCO_3	(mEq/l)		33	24	31	20	19	20			
	art.Sat.	(%)		99	99	99	99	99	98			
	ven.Sat.	(%)		77	73	79	68	56	64			
Flow		(cc)			2450	3300	3100	3300				
		(cc/Kg)			35	47	44	47				
O_2Cons.(ml/min/m^2BSA)					39	49	71	99				
Hematocrit			48	43	26	27	27	26	36		39	
Hemoglobin		(gm%)	16.6	14.9	9.0	9.3	9.3	9.0	12.4		13.3	
Lactate & Saline		(cc)				3100						
Urine Volume		(cc)		25		145			130		570	
Urine Potassium		(mEq)									58	
Net Fluid Change		(cc)									2200	
Red Cell Volume		(cc)	1546								1400	
Plasma Volume		(cc)	2600								2342	

Name - R.B.
Case No - 20
Age - 51
Sex - M

Ht- 72"
Wt- 189 lbs.
- 86 Kg.
Body Surface Area (m^2) - 2.08 M^2

Operation - Aortic Valve Replacement

Perf.Time - 92 mins.
Group - II

Meds.- Diuretics pre-op, Digitalis (post-op)
Arrhythmias- nil

Hypothermia - No.

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.4	4.9	4.9	5.0	5.0	5.2	4.6	4.0	3.6
Gases	art.pO_2	(mmHg)		160	176	265	174	260	87		
	ven.pO_2	(mmHg)		31	28	31	38	39	44		
	art.pCO_2	(mmHg)		34	29	29	27	24	39		
	ven.pCO_2	(mmHg)		38	45	40	31	31	42.5		
	pH			7.44	7.43	7.49	7.52	7.60	7.32		
	art.HCO_3	(mEq/l)		23	20	23	23	24	19		
	act.HCO_3	(mEq/l)		22	19	21	21	22	19		
	art.Sat.	(%)		99	99	99	99	99	95		
	ven.Sat.	(%)		57	49	57	64	66	73		
Flow		(cc)			3300	3400	3700	3700			
		(cc/Kg)			38	40	43	43			
O_2Cons.(ml/min/m^2BSA)					72	73	62	68			
Hematocrit			49	40	24	26	26	28	37	42	
Hemoglobin		(gm%)	16.7	13.6	8.2	8.9	8.9	9.6	12.6	14.3	
Lactate & Saline		(cc)				2070					
Urine Volume		(cc)		0		80			40	700	
Urine Potassium		(mEq)								149	
Net Fluid Change		(cc)								1780	
Red Cell Volume		(cc)	1723							1480	
Plasma Volume		(cc)	2745							2760	

Name - H.W.
Case No - 21
Age - 68
Sex - M

Ht - 68"
Wt - 152 lbs.
-69 Kg.
Body Surface Area (m^2) - 1.81 M^2

Operation - Repair of torn chordae

Perf.Time - 37 mins.
Group - I

Meds. - Digitalis (post-op)

Arrhythmias - nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K	(mEq/l)	4.0	5.0	5.4	6.1			6.0	4.8	4.2	3.5
	art.pO_2	(mmHg)		59	290	380			58			
	ven.pO_2	(mmHg)		38	38	33.5			36			
	art.pCO_2	(mmHg)		36	22	20			30			
	ven.pCO_2	(mmHg)		44	27.5	31.5			35			
	pH			7.41	7.53	7.58			7.43			
	art.HCO_3	(mEq/l)		23	21	22			21			
	act.HCO_3	(mEq/l)		22	18	18			19			
	art.Sat.	(%)		89	99	99			89			
	ven.Sat.	(%)		69	78	68			68			
Flow		(cc)			3200	3500						
		(cc/Kg)										
O_2Cons.(ml/min/m^2BSA)					54	82						
					54	82						
Hematocrit			51	42	28	30			33	42		
Hemoglobin		(gm%)	16.3	13.6	9.0	10.0			10.9	13.8		
Lactate & Saline		(cc)				1580						
Urine Volume		(cc)		30		10			170	950		
Urine Potassium		(mEq)								88		
Net Fluid Change		(cc)								782		
Red Cell Volume		(cc)	1633							1226		
Plasma Volume		(cc)	2355							2790		

Name - P.R.
Case No - 22
Age - 43
Sex - M

Ht- 67"
Wt- 182 lbs.
- 82 Kg.
Body Surface Area (m^2) - 1.94 M^2

Operation - Aortic Valve Replacement

Perf.Time - 87 mins.
Group - I I

Meds.- Digitalis (pre-op, post-op)
Arrhythmias- Extrasystoles (post-op)
Hypothermia - No

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K (mEq/l)	3.8	4.8	4.8	4.7	4.7	5.0	4.5	4.2	4.0
	art.pO_2 (mmHg)		80	89	230	290	375	110		
	ven.pO_2 (mmHg)		24	30	37	37	36	30		
	art.pCO_2 (mmHg)		33	39	34	31	29	42		
	ven.pCO_2 (mmHg)		30	36	37.5	35.5	35.5	44		
	pH		7.44	7.37	7.42	7.47	7.49	7.37		
	art.HCO_3 (mEq/l)		22	22	22	23	23	23		
	act.HCO_3 (mEq/l)		21	21	21	22	22	24		
	art.Sat. (%)		95	96	99	99	99	99		
	ven.Sat. (%)		53	58	72	73	72	53		
Flow	(cc)			3500	3700	3700	3700			
	(cc/Kg)			43	45	45	45			
O_2Cons.(ml/min/m^2BSA)				74	69	71	79			
Hematocrit		46	48	29	31	32	32	42	44	
Hemoglobin	(gm%)	16.4	17.1	10.3	11.0	11.4	11.4	15.0	15.6	
Lactate & Saline	(cc)				2250					
Urine Volume	(cc)		20		210			185	560	
Urine Potassium	(mEq)								61	
Net Fluid Change	(cc)								1635	
Red Cell Volume	(cc)	1955							1950	
Plasma Volume	(cc)	3100							3260	

Name - W.Z.
Case No - 23
Age - 52
Sex - M

Ht- 65"
Wt- 132 lbs.
- 60 Kg.
Body Surface Area (m^2) - 1.66 M^2

Operation - Mitral Valve & Tricuspid Replacements

Perf.Time - 99 mins.
Group - II

Meds. - Diuretics; Digitalis (pre-op, post-op)
Arrhythmias - nil

Hypothermia - No

			Pre		Bypass			Post		Post-op	
		Pre-op	Bypass	1	2	3	4	Bypass	Day 1	Day 5	
Blood	K (mEq/l)	3.5	4.5	4.7	4.8	4.6	4.5	3.7	3.6	4.1	4.3
Gases	art.pO_2 (mmHg)		85	136	305	390	430	83			
	ven.pO_2 (mmHg)		36	36	37	30	23	35			
	art.pCO_2 (mmHg)		39	37	32	38	26	50			
	ven.pCO_2 (mmHg)		57	45	39	40	32.5	55			
	pH		7.38	7.42	7.53	7.57	7.64	7.31			
	art.HCO_3 (mEq/l)		23	24	27	34	32	23			
	act.HCO_3 (mEq/l)		22	23	25	34	30	24			
	art.Sat. (%)		95	99	99	99	99	99			
	ven.Sat. (%)		62	62	71	64	50	44			
Flow	(cc)			3500	3600	3600	2300				
	(cc/Kg)			58	60	60	38				
O_2Cons.(ml/min/m^2BSA)				86	89	63	106				
Hematocrit		50	50	32	38	30	31	37		42	
Hemoglobin	(gm%)	16.2	16.1	10.3	12.3	9.7	10.0	12		13.6	
Lactate & Saline	(cc)				2500						
Urine Volume	(cc)		15		110			55		700	
Urine Potassium	(mEq)									46	
Net Fluid Change	(cc)									1582	
Red Cell Volume	(cc)	1900								1605	
Plasma Volume	(cc)	2010								2210	

Name - W.G. Ht- 63"
Case No - 24 Wt- 147 lbs.
Age - 52 - 67 Kg.
Sex - M Body Surface Area (m^2) - 1.67 M^2

Operation - Aortic Valve Replacement

Perf.Time - 81 mins.
Group - II

Meds. - Nil

Arrhythmias - nil

Hypothermia - No

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K	(mEq/l)	3.9	4.6	4.9	5.1	5.3	5.7	4.7	4.5	3.2
	art.pO_2	(mmHg)		120	320	315	300	305	197		
	ven.pO_2	(mmHg)		37	34	37	30	29	31		
	art.pCO_2	(mmHg)		31	33	35	24	21	38		
	ven.pCO_2	(mmHg)		35	38	42	38	41	44		
	pH			7.45	7.42	7.44	7.56	7.56	7.43		
	art.HCO_3	(mEq/l)		26	23	24	24	24	25		
	act.HCO_3	(mEq/l)		25	21	23	21	23	24		
	art.Sat.	(%)		99	99	99	99	99	99		
	ven.Sat.	(%)		70	62	68	57	58	56		
Flow		(cc)			3200	3300	3300	3300			
		(cc/Kg)			47	49	49	49			
O_2Cons.(ml/min/m^2BSA)					77	74	81	98			
Hematocrit			44	39	26	27	27	28	40		
Hemoglobin		(gm%)	14.1	12.5	8.3	8.6	8.6	9.0	13.9		
Lactate & Saline		(cc)				1540					
Urine Volume		(cc)		20		40			10	825	
Urine Potassium		(mEq)								93	
Net Fluid Change		(cc)								1446	
Red Cell Volume		(cc)	1278							1410	
Plasma Volume		(cc)	2540							2735	

Name - V.M.
Case No - 25
Age - 51
Sex - M

Ht- 73"
Wt- 151 lbs.
- 69 Kg.
Body Surface Area (m^2) - 1.90 M^2

Operation - Aortic Valve Replacement

Perf.Time - 89 mins.
Group - II

Meds.- Digitalis (post-op)

Arrhythmias- nil

Hypothermia - 31° C

			Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood	K	(mEq/l)	4.7	4.5	3.9	4.5	4.5	4.3	4.3	3.8	4.0	4.3
Gases	art.pO_2	(mmHg)		98	313	370	325	104	96			
	ven.pO_2	(mmHg)		26	41	42	30	28	23			
	art.pCO_2	(mmHg)		34	34	34	25	22	44			
	ven.pCO_2	(mmHg)		34.5	34.5	34	34	31	51			
	pH			7.37	7.39	7.43	7.57	7.50	7.38			
	art.HCO_3	(mEq/l)		20	21	23	26	21	24			
	act.HCO_3	(mEq/l)		20	21	22	22	24	25			
	art.Sat.	(%)		97	99	99	99	99	96			
	ven.Sat.	(%)		44	71	74	65	60	35			
Flow		(cc)			3500	3800	3800	3800				
		(cc/Kg)			48	52	52	52				
O_2Cons.(ml/min/m^2BSA)					61	66	70	77				
Hematocrit			43	42	27	27	26	31	43		44	
Hemoglobin		(gm%)	14.1	13.7	8.9	8.9	8.5	10.1	14.1		14.5	
Lactate & Saline		(cc)				2100						
Urine Volume		(cc)		20		130			70		550	
Urine Potassium		(mEq)									47	
Net Fluid Change		(cc)									1990	
Red Cell Volume		(cc)	1570								1810	
Plasma Volume		(cc)	3123								3130	

Name - S.P. Ht- 75"
Case No - 26 Wt- 192 lbs.
Age - 44 - 86 Kg.
Sex - M Body Surface Area (m^2) - 2.16 M^2

Operation - Mitral Valve & Aortic Valve Replacements & Tricuspid Annuloplasty
Perf.Time - 148 mins.
Group - III

Meds. - Diuretics; Digitalis, (pre-op)
Arrhythmias- Atrial & Ventricular Fibrillation
Hypothermia- No

		Pre-op	Pre Bypass	Bypass 1	Bypass 2	Bypass 3	Bypass 4	Post Bypass	Post-op Day 1	Post-op Day 5
Blood Gases	K (mEq/l)	3.7	4.1	4.4	4.5	4.4	4.5	5.8	5.4	3.7	3.6
	art.pO_2 (mmHg)		64	116	320	330	500	63			
	ven.pO_2 (mmHg)		33	36	40	42	44	35			
	art.pCO_2 (mmHg)		27	29	22	39	34	45			
	ven.pCO_2 (mmHg)		30	36	27.5	35.5	35.5	44			
	pH		7.39	7.50	7.62	7.43	7.43	7.32			
	art.HCO_3 (mEq/l)		20	24	25	25	23	21			
	act.HCO_3 (mEq/l)		16	22	22	25	22	22			
	art.Sat. (%)		91	99	99	99	99	98			
	ven.Sat. (%)		56	69	73	77	79	57			
Flow	(cc)			3600	4100	4100	4100				
	(cc/Kg)			41	47	47	47				
O_2Cons.(ml/min/m^2BSA)				59	70	62	69				
Hematocrit		44	38	32	31	32	34	41			
Hemoglobin	(gm%)	14.5	12.5	10.5	10.2	10.5	11.2	13.5			
Lactate & Saline	(cc)				2700						
Urine Volume	(cc)		0		150			85	800		
Urine Potassium	(mEq)								91		
Net Fluid Change	(cc)								2465		
Red Cell Volume	(cc)	2590							2620		
Plasma Volume	(cc)	4540							3840		

APPENDIX 7

COMPUTER PROGRAM FORMAT

FOR CALCULATION OF

CORRELATION COEFFICIENTS (51)

```
 1. Let A = Ø.Ø
 2. Let A1= Ø.Ø
 3. Let B = Ø.Ø
 4. Let B1= Ø.Ø
 5. Let C = Ø.Ø
 6. Let C2= Ø.Ø
 7. Let D = Ø.Ø
 8. Let F = Ø.Ø
 9. Let F1= Ø.Ø
10. Let G = Ø.Ø
11. Let H = Ø.Ø
12. Let J = Ø.Ø
14. Let M = Ø.Ø
15. Let N = Ø.Ø
16. Let Q = Ø.Ø
17. Let R = Ø.Ø
18. Let T = Ø.Ø
19. Let Z = Ø.Ø
20. Dim X(11Ø), Y(11Ø)
21. Print "How many values of X and
      Y do you have?"
22. Input N
23. For I = 1 to N
24. Read X(I)
25. Next I
26. For I = 1 to N
27. Read Y(I)
28. Next I
29. For I = 1 to N
30. Let A1 = X(I)*Y(I)
31. Let A = A + A1
32. Next I
33. For I = 1 to N
34. Let B2 = B2 + X(I)
35. Next I
36. Let B = B2/N
37. For I = 1 to N
38. Let C2 = C2 + Y(I)
39. Next I
40. Let C = C2/N
41. Let D = N*B*C
42. Let T = A - D
43. For I = 1 to N
44. Let F1 = X(I)↑2
45. Let F = F + F1
46. Next I
47. Let G = N*B↑2
48. Let H = F - G
49. For I = 1 to N
50. Let J1 = Y(I)↑2
51. Let J = J1 + J
52. Next I
53. Let M = N*C↑2
54. Let Q = J - M
55. Let Z = (H*Q)↑Ø.5
56. Let R = T/Z
57. Print "The Coefficient of
      Correlations is"; R
```

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